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A HANDBOOK

OF

MEDICAL DIAGNOSIS

FOR STUDENTS.

BY

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TO

JAMES NEVINS HYDE, A.M., M.D.,

WHOSE

EXAMPLE, COUNSEL AND FRIENDSHIP

HAVE BEEN TO ME, DURING

UNDERGRADUATE AND PROFESSIONAL LIFE,

OF THE

GREATEST VALUE.

32071

PREFACE.

THIS book is intended chiefly for students. The aim has been to bring together into compact form the main facts that assist one in making a diagnosis; those to be derived from subjective and objective examinations, together with those derived from chemistry, bacteriology, and microscopy. The attempt has been made to combine the more important and essential parts of the text-books devoted to chemical and microscopical diagnosis, those devoted solely to physical diagnosis, and those paying special attention to symptomatology. To this end free use has been made of standard treatises on Medicine, with its specialties, and of the now numerous excellent text-books on Physical Diagnosis, Medical Diagnosis, and Clinical Medicine.

It is hoped that the volume will be more than a quiz-compend; that it will stimulate the undergraduate to further study in larger works and in good current medical literature; to more careful and thorough clinical observation, and to original investigation.

JAMES B. HERRICK.

751 WARREN AVE., CHICAGO.
September, 1895.

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HANDBOOK OF MEDICAL DIAGNOSIS.

INTRODUCTION.

GENERAL CONSIDERATIONS.

THE most important question the physician is called upon to answer when he is at the bedside of his patient is not what is the cause of the disease or its proper treatment, but what *is* the disease. What is the matter? The mastery of the diagnosis is half the battle. Treatment follows naturally when the ailment is known, and especially if an operating causal factor can be removed. Now, just as treatment is either rational or empirical, so diagnosis may in a sense be rational or empirical—rational where the physician in diagnosing typhoid fever or pneumonia has a clear conception of the causative agents of the disease, the pathological processes thereby excited and the morbid changes wrought in various organs of the body with consequent alteration of function; empirical where, without any deep insight into the changes produced, he recognizes by certain clinical features the likeness of the case in hand to the case of his text-book, and so names his disease, and prescribes according to name and not the actual condition of the patient before him.

The ability to make an accurate and, at the same time, rapid diagnosis is, in a measure, a gift, but in much larger degree the result of careful training and experience. Students are prone to look upon the diagnoses of their preceptors or clinical instructors as made by intuition. But the accurate diagnosis is made as the result of observation and reasoning, though a trained eye and mind may reach the result quickly by passing rapidly over ground that the beginner treads with hesitating caution. As the pianist, by daily practice of scales and studies, learns to play at sight music that in his early years would have required hours of patient study for its correct performance, so the diagnostician by going repeatedly over the same ground soon learns to read his cases at sight. But it is not intuition; it is the result of careful training. The eye that at first analyzed the scarlatinal rash minutely and in detail, and made careful inquiry into the history of the case, mode of onset, exposure to the disease, coexistence of sore-throat, high fever, etc , now at a glance recognizes the eruption, with the finger on the rapid pulse detects the heat of the skin, and makes a so-called intuitive diagnosis. Yet there is a process of reasoning here, unconscious though the reasoner himself may be of it. But practice has enabled this physician to reach his conclusions by rapid strides instead of by a slow and painstaking process.

There is no such thing as an intuitive diagnosis.

EXAMINATION OF THE PATIENT.

It is well, therefore, for the sake of the drill and for the thoroughness it inculcates that the student and practitioner in their examination of patients follow some definite method. In this way important facts that otherwise might be slighted are not overlooked. It is safe to say that more mistakes in diagnosis are made through lack of carefulness in examination than through ignorance.

An examination includes both a history of the patient up to the time he presents himself to the physician and a direct inspection and exploration of the patient by the physician. The former, often spoken of as the *anamnesis*, is subjective; the latter, objective, is the *status præsens*. In practice it is often impossible or impracticable to follow literally a definite order of questioning and examination, yet every physician should have some scheme which in perplexing cases, especially those of a chronic character, he follows closely, and to which he adheres as rigidly as possible in every instance. The following plan will, perhaps, serve as a model which can be modified to suit individual taste. It is slightly altered from that of the first medical clinic at Berlin:

Name, age, social condition. Date of the examination.

ANAMNESIS.

1. *Hereditary History.*
2. *Habits and Occupation.*
3. *Previous History.*

(*a*) Childhood History; (*b*) Menstruation; (*c*) Pregnancies; (*d*) Preceding illnesses or injuries.

4. *Present Illness.*

(*a*) Time of attack, apparent cause, prodromes, initial symptoms, course of disease, previous treatment, etc.

(*b*) Previous complaints, *e. g.*, appetite, pain, cough, urine, sleep, etc.

STATUS PRÆSENS.

A. General symptoms and signs.
  1. General appearance, stature, musculature, bony structure, panniculus adiposus.
  2. Position in bed or manner of moving.
  3. Countenance—expression, color, etc.
  4. Skin—color, œdema, scars, moisture, eruptions.
  5. Pulse—frequency, rhythm, etc. Condition of artery.
  6. Temperature.
  7. Respiration—frequency and type.
  8. Sensations and complaints of patient—pain, sleep, weakness, urine, etc.

B. Special symptoms and signs.
  1. Nervous System—Sensorium, headache, dizziness, sleep, tremor, sensory and motor disturbances, etc.
  2. Digestive System—Tongue, throat, stomach, bowels, etc., examination of stomach contents.
  3. Respiratory System, including physical examination of lungs and of sputum.
  4. Circulatory System—Physical examination of heart and vessels, examination of blood.
  5. Urine—Amount, specific gravity, reaction, color, chemical and microscopic examination.

Diagnosis.
Treatment.
Subsequent observations.

A few words in explanation of some of these points and to show in what manner they may have a direct bearing upon the establishment of a diagnosis may not come amiss.

Hereditary History. Whatever heredity may really mean, we know that there is a tendency for disease that affected the progenitors to appear in the offspring. This is true not alone of tubercular and cancerous diseases, but of rheumatism, chronic nephritis, various nervous diseases, and early degenerative changes in the bloodvessels.

Habits and Occupation. Alcoholism, excessive use of tobacco, sedentary life, may give us the clew to an otherwise doubtful case of liver or heart disease or of constipation. The worker in lead may have his symptoms explained by our knowledge of his occupation. So the case of emphysema or of phthisis is made clearer to us by a knowledge of the fact that our patient is a blower of a wind instrument or is a stone mason.

Previous History. The history of preceding scarlet fever or rheumatism puts us on the lookout for renal or cardiac sequelæ. A previous suppurating focus may explain a septic trouble in the liver, heart, or pleural cavity. The fact that a child has had previously scarlet fever is *prima facie* evidence against the second attack, as the preceding history of erysipelas or pneumonia or tonsillitis would favor the diagnosis of a recurrence. Repeated miscarriages may be all the evidence we have of constitutional syphilis.

Present Illness. No mistake is oftener made by the beginner than to forget to inquire minutely as to the time when the patient first became ill. It is very chagrinning after you have made up your mind that you have to deal with an acute inflammation of, we will say the stomach, to learn that the patient has been a sufferer for months or years, and that you are but the last of many physicians whom he has consulted. The time of the onset of the disease usually definitely puts the affection at once into one or the other of the two great classes of acute or chronic maladies. While we have to make much allowance for the statements of patients as to the cause of the disease, its course, and the treatment adopted by themselves or by some other doctor, and the effects of such treatment, still much light may be thrown upon the diagnosis by intelligent patients. A knowledge, for instance that a headache improved under iodides, or that quinine in large and repeated doses has failed to check recurring chills, may be the fact leading to the decision of syphilis or suppuration.

General Symptoms and Signs. Much is to be learned from the general appearance of the patient. The well-nourished woman with florid cheeks, whose chief complaint is of the stomach, which has been a source of trouble for five years, is one in whom we exclude almost positively carcinoma. In the cachectic individual, on the contrary, pale, emaciated, weak, with some chronic ailment, we are on the lookout for carcinoma, tuberculosis, leukæmia, diabetes, etc. The position in bed should be noted. With wasting diseases, *e. g.*. typhoid, where the system is profoundly depressed, we are prone to find our patient on the back, chin dropped upon the chest, head slipped away from the pillow, knees flexed, the body fallen together, as it has been expressed, *i. e.*, in the passive dorsal decubitus. In the active dorsal decubitus the head is square upon the pillow, moved freely to observe the actions of attendants, the knees straight. Such a position we might have in a case of sthenic pneumonia. A lateral decubitus speaks for a one-sided affection of the chest, as fluid or air in the pleural cavity. The sitting posture constantly maintained points to embarrassed heart action and consequent dyspnœa, to cardiac dilatation, pressure of fluids in the chest, or an asthmatic paroxysm. Attention need only be called to the gait of the patient to show its importance in such cases as spinal cord diseases, paralyses, injuries or diseases of the joints of the lower extremities.

Countenance, Color of Skin, etc. The experienced physician often learns much by a mere glance at the face of his patient. The pale face and lips, the anxious look, the restless eye tell, even before the finger is put to the pulse, of loss of blood. The pinched nose, sunken eyes, ashy-colored countenance, with, perhaps, beads of sweat upon it, speak of suffering or of pronounced sepsis, as in peritonitis. The pale face of chlorosis, the puffy, waxy countenance of Bright's disease are not less characteristic than the bronzed hue of Addison's disease or the yellow tint of jaundice. The countenance of fever with its flushed cheek and hot, dry skin, often betrays by its expression something as to the character of the illness. The dull, listless countenance of the typhoid, who, unmindful of the flies that light upon him or of the gaze of the visitor, lies with mouth open, displaying the teeth and tongue dry and covered with sordes, is very different from that of the pneumonic, with flushed cheek, restless eye, face expressive of pain, and who asks for relief the moment the doctor is within hearing distance. The bloated face of the alcoholic, the drooping, often trembling lid of the hysterical, the immobility of one side of the face in the sufferer from facial paralysis, all tell their tales to the eye of the careful observer. And the scar on the face, as on other portions of the body, may be as significant of a previously existing syphilitic lesion as the relic of the initial sore or the notched teeth of the child hereditarily tainted.

Pulse. Much is learned by an examination of the pulse. Ordinarily the radial pulse at the wrist is examined. Where there is tendinous twitching that interferes with the examination of this artery, or where the radial pulse is very weak, or where, because of the jerking of the arms, as in nervous, resisting children, the radial pulse cannot be examined, the carotid, the temporal or femoral pulse may be substituted.

There should always be noted the rhythm, the fulness and tension. An irregular pulse is one of graver significance than an intermittent one; in fact, some persons, even in conditions of health, have the pulse occasionally skip a beat. Increased arterial tension, as evidenced by the pulse, may be of great value in determining the existence of diseased bloodvessels, of cardiac hypertrophy, or of renal disease. A comparison of the fulness of the pulse and its strength, with the strength of the heart-beat, may be of value, as, for instance, in certain cases of valvular disease, where an hypertrophied heart with a full, strong beat may still, because of a valvular leak, be attended by a weak and feeble pulse. The two radial pulses should be compared in cases of suspected thoracic aneurism.

Excitement may increase the frequency of the pulse-beat. It is regularly increased in fevers. It is generally the rule that the higher the temperature the more rapid the pulse. To this rule there are some exceptions, as, for instance, in typhoid fever a quite high tem erature may be attended by a pulse but slightly increased in frequency. In scarlet fever, on the contrary, a slight rise in temperature is usually accompanied by a very rapid pulse, a pulse of 130 or 140 being not infrequent in this disease. A very rapid pulse in patients who are up and about should call attention to the possibility of organic or functional heart disease, of exophthalmic goitre, or of pulmonary tuberculosis. Not infrequently patients with pulmonary tuberculosis do a day's work with a pulse averaging 120 to 130. A slow pulse is natural with some people. During convalescence from acute infectious diseases bradycardia may be present. Cerebral compression often produces a slowing of the pulse-rate.

Temperature. The only sure way of determining the body temperature is by the use of the thermometer. Fairly good results can, however, by practice, be obtained by feeling of the skin and observing the pulse-rate. The ordinary clinical thermometer, if placed under the tongue, should be kept there at least three minutes; if in the axilla or the groin, the latter being an excellent place in children, at least five minutes; in the rectum or vagina, two or three minutes usually suffice. In the axilla and groin the temperature is about one-half degree lower than under the tongue, and about one-half degree lower under the tongue than in the rectum. Even in health the evening temperature is usually a little higher than the morning. In children the temperature can rapidly rise to quite a high point—103° or 104° on comparatively slight provocation. The same is true to a less extent in nervous people.

Fever is usually indicated when a patient has a chill or complains of chilliness, or of cold, creeping sensations. The physician cannot always credit the statements of patients as to the existence of fever. The skin feels oftentimes to the patient hot and burning, when in reality there is no increase in the temperature of the body. The general symptoms of fever are practically the same, whatever the cause of the fever. There is usually headache, general malaise, loss of appetite, a feeling of languor, cold, creepy feelings, succeeded by sensations of heat.

Respiration. The normal respiration rate for an adult is about 18 per minute. With an elevation of temperature respiration is usually increased in rapidity. Marked increase in the rate of breathing usually calls attention to the heart or lungs as the source of the trouble. The most rapid respiration is seen in children, where there is extensive involvement of the smaller capillary tubes, as in lobular pneumonia. With pleurisy the respiration is catchy. With a large intrathoracic effusion there is oftentimes lateral decubitus with embarrassed respiration.

Difficulty in breathing is known as dyspnœa. Where the patient is obliged to sit up in order to breathe, the condition is known as orthopnœa. Great difficulty in expiration is seen in emphysema and in asthma. In the latter condition the respiration is wheezing. With laryngeal obstruction, as in diphtheric laryngitis, there may be difficulty in inspiration and in expiration. Inspection of the soft parts is of value in determining the degree to which the lungs are filled with air. Where there is marked obstruction to the entrance of air, the soft parts above the clavicle, in the epigastrium, and also the lower ribs are seen to sink in, instead of to expand with inspiration.

Slow respiration is common in compression of the brain and in opium-narcosis. By Cheyne-Stokes respiration is meant a peculiar form of breathing that is seen in connection with nervous troubles and brain lesions. The patient breathes at first quietly and slowly, then more and more rapidly, takes a deep inspiration, and then stops for a few seconds, then begins again to breathe slowly, and increase in rapidity as before.

Sensations and Complaints of Patients. It requires a great deal of tact to unravel from the tangled story that many patients give to us of their ailment the facts that are essential. It is sometimes wise not to allow a patient to tell his story as he wishes, but to insist upon his merely answering questions that are asked. This frequently saves time and prevents the physician from becoming confused as to the true nature of the malady. Yet, on the other hand, the physician may make a mistake in asking questions leading in the direction in which his preconceived notion as to what ails the patient points. It is oftentimes wise, therefore, to allow the patient to tell his story in his own way without interruption, even though valuable time is consumed with the rehearsal of details that are unimportant. The physician will in this way frequently avoid some serious error in diagnosis, as his attention will be called to some fact that he might not have detected had he merely acted the part of a cross-examiner.

The complaint of patients as to pain and suffering are frequently unconsciously exaggerated; and, again, there are those who minimize their sufferings. The nervous, hysterical girl really feels and suffers more pain, and makes greater complaint of her sufferings, than does the strong, robust, stoical man. Malingerers are frequently met with, particularly where examinations are made for life insurance, for pensions, etc. It is a fact of common observation, too, that patients who dread the disclosure of the existence of some serious disease as cancer, tuberculosis, or Bright's disease, often try to conceal every fact that would aid the physician to make the diagnosis. The patient frequently has an entirely wrong notion as to the organ that is really diseased. Unless the physician be wary he will be put upon the wrong track by importance attached by the patient to symptoms referable to some organ other than the one primarily affected. Very commonly patients with heart disease or with pulmonary tuberculosis complain of the stomach. The diabetic may make complaint of the skin disease or of his neuralgia. The patient with nephritis is bothered greatly with dyspnœa; the chlorotic suffers from headache and dizziness; the tabetic thinks he has rheumatism, etc.

PHYSICAL EXAMINATION.

We all make many mistakes because we are not more careful and thorough in our physical examinations. We fail to make an examination, perhaps, because we are in a hurry. Or motives of modesty, delicacy, or fastidiousness lead us to neglect the vaginal or rectal examination or the examination of the sputum. Patients sometimes think these examinations unnecessary, and we readily yield to their judgment. Yet, because it is right as well as from motives of policy, it will be found wise always on the first visit to a patient to make a careful physical examination. Patients in the end learn to like thoroughness; they learn to respect the man who makes a careful and complete physical examination.

Some systematic order of procedure should always be followed in making a physical examination, at least by the beginner, as, for instance, an examination first of the respiratory tract; then the heart and bloodvessels, next the alimentary tract, then the nervous system, etc. Or perhaps oftener, an examination, in order, of the body, from above downward, first of the head, then the thorax and abdomen, and lastly the extremities. It is wise with all patients, particularly office patients, to keep case-records showing the result of examinations made at various times. A little practice enables the physician to make these records very rapidly.

DIAGNOSIS.

Where possible a diagnosis should be made positively. Yet an honest confession of ignorance is often the best, as it is the only honorable policy. Intelligent people are willing to wait until a physician has had time to observe the patient and until he can arrive at a positive conclusion. It is only the ignorant who expect a diagnosis to be made at first sight in every instance. People are beginning to learn that it is necessary in some cases to examine carefully all the organs of the body, together with the secretions and excretions. They are willing to wait, therefore, until a urinalysis has been made, until, perhaps, the sputum has been examined, or until the suspicious deposit in the throat has been subjected to bacteriological examination. More credit often comes to the physician who makes a correct diagnosis, and from that a correct prognosis, than to the physician who cures all cases.

THE INFECTIOUS DISEASES.

THE **acute infectious diseases** are parasitic in their origin, many of them being at the same time highly contagious. The group often spoken of as the **acute eruptive fevers**, and including scarlet fever, measles, röthlen, chicken-pox, smallpox, typhoid fever, typhus fever, has certain common characteristics: (1) a period of incubation, from the entrance of the micro-organism into the human body to the manifestation of its influence by (2) the prodromal symptoms; (3) fever running a definite course and self-limited; (4) an eruption; (5) protection against a second attack usually afforded by the first.

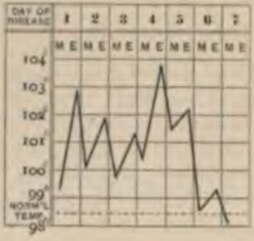
Symptoms of the Febrile Condition. The general symptoms of the febrile condition, no matter what the infective agent, are to be remembered as common not alone to this group of diseases but to all fevers. There are chilly sensations followed by a sense of heat, headache, backache, often general aches and pains, "pain in the bones;" the patient feels tired, languid, has "general malaise;" the appetite is lost, the tongue usually coated; the skin is flushed, the pulse is increased in frequency, and at first, except with profound infections, is full and strong.

MEASLES.

After a period of incubation of from ten to fourteen days the symptoms of an ordinary "cold in the head" are manifest, watery eyes, inflamed nose and throat, slight cough, with some fever. This continues for four days, when the rash begins to appear; and until the eruption is fully developed, for forty-eight hours, the temperature remains near 103°, falling suddenly when the rash is completely out. A convulsion may occur as the rash begins to appear.

Temperature. During the prodromal stage of four days the temperature is rarely higher than 102°, and often on the second or third day is below 100°. As the rash appears it rises to 103° or even higher, remaining so for twenty-four to sixty hours, and then in uncomplicated cases dropping to nearly normal by crisis. Temperature after the seventh day of measles can be looked upon as due to a complication or to an unusually severe intoxication of the system. The pulse of measles is generally full and strong, not so rapid as in scarlet fever.

FIG. 1.



Temperature in measles.

Rash. On the fourth day can be seen near the edge of the hair, on the forehead and back of the ears, small, discrete, reddish papules, soon slightly elevated and rapidly increasing in number, and extending downward over the entire body. Many of these coalesce, forming dark reddish blotches, especially noticeable on the swollen face and chest. It takes two days for the rash to reach the feet. It remains out for about forty-eight hours, disappearing in the same order in which it developed. A yellowish-brown discoloration, persisting for several days, may mark the site of a previous papule. Desquamation occurs in fine furfuraceous scales, often scarcely noticed where the daily sponge bath has been employed.

The crescentic outline of the papules can rarely be made out, and is of slight value in diagnosis.

Other symptoms are the coryza, the cough, and occasionally the diarrhœa. In fact, the mucous membranes of the eye, nose, larynx, bronchi, and often of the stomach and intestines, are in a state of acute catarrhal inflammation and give the ordinary manifestations of such trouble. The bronchitis is oftentimes marked, moist and dry râles being abundant.

Varieties. Ordinarily when uncomplicated, a trifling disease, measles may assume from the start a serious aspect. In these cases of so-called malignant measles not only is the temperature usually higher and the pulse more rapid, but the nervous system is profoundly shocked. Delirium or stupor may occur early, the rash is of a purplish tint, petechiæ are seen, and hemorrhages sometimes occur from the mucous membranes. The patient may die in this typhoid state. Measles is often of the severe type in adults.

Complications. The complications of measles are chiefly exaggerations of the already existing inflammations of the mucous membranes. Severe conjunctivitis or keratitis, the latter often of the phlyctenular type, may occur early or late during the course of the disease. Extension of the naso-pharyngeal catarrh to the middle ear, by way of the Eustachian tube, not infrequently occurs.

A membranous laryngitis, at times without evidence of diphtheritic deposits in the fauces, and again with faucial diphtheria, is one of the dreaded complications. It seems at times as though this membranous deposit crept up from below, covering first the walls of the bronchi and trachea, and lastly the vocal cords, rather than by the usual method of involving first the throat and then the larynx and trachea. Difficult breathing, hoarseness, croupy cough, livid lips, yet pallid face, retraction of the soft parts, give evidence of the obstruction of the glottis.

When, after the subsidence of the fever and the disappearance of the rash, there is a sudden increase in temperature, rapid, panting respiration, quickened pulse-rate, we must examine closely for the subcrepitant râle of capillary bronchitis and for the small areas of consolidation of lobular pneumonia that almost invariably accompany it. At times the capillary bronchitis is very insidious in its approach. Lobar pneumonia is also occasionally found after measles. These three diseases, laryngitis, capillary bronchitis, pneumonia, coming as complications, make measles a prolific cause of death in children under five years of age.

Enteritis evidenced by the pronounced diarrhœa is at times annoying.

Among the sequelæ of measles should be mentioned chronic conjunctivitis, otitis media, bronchitis A latent tuberculosis is at times lighted up by the disease, and adenitis, often tubercular, is frequently reported as having started at the time the child was afflicted with measles.

GERMAN MEASLES.

German measles, röthlen or rubella, is an eruptive disease now generally regarded as entirely distinct from measles and scarlet fever. Though no specific causal micro-organism has been isolated, the disease from its peculiar clinical picture, and from the immunity afforded by one attack against a second, though not against measles or scarlet fever, is to be looked upon as a distinct entity, neither a modification of measles or scarlet fever, nor a hybrid of both.

The period of incubation of about ten days is followed by a slight prodromal coryza and temperature usually overlooked. The first evidence of the disease is often the appearance of the rash. During an epidemic of German measles children are often sent home by teachers, and are only convinced that they are ill by looking in the mirror and noticing the rash fully developed on the face. The rash appears rapidly, and often at about the same time, on the face, trunk, and extremities, though with a tendency to begin on the neck and face. It consists of pale, reddish, discrete, very slightly elevated papules, seldom as large as those of measles, but in the aggregate, because of their failure to become confluent, giving, as we glance at the skin of the patient, the impression that they are much smaller and sprinkled indiscriminately over the body. The eruption persists for one to four days and gradually disappears. There has not been uniformly noted desquamation, though a fine branny scale usually is shed after the rash fades. The fever is rarely higher than 101° or 102°, and at times appears to be wanting. The cervical glands, especially those just back of the sterno-mastoid, are enlarged and sometimes slightly tender. So, too, the occipital, auricular, and submaxillary glands are often plainly noticeable. The coryza and cough are rarely so marked as in measles, though more complaint is made of soreness of the throat. Complications are exceedingly rare, and are such as would be caused by exposure, *i. e.*, exaggerations of the existing catarrhal inflammation of the mucous membranes. The fatal cases are usually those in which pneumonia and bronchitis have occurred. Occasionally, as in even the mildest of the infectious diseases, this disease assumes a severe type, though fatal results are practically unknown in private practice. In hospitals and among weak and sickly children, with poor hygienic surroundings, the disease has had a mortality as high as nine per cent.[1]

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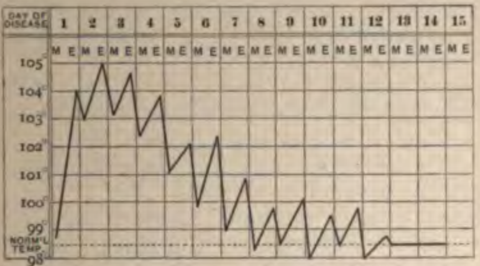
[1] Vide. The very complete article, "Rubella," by W. A. Edwards—Keating's Cyclopædia of Diseases of Children, Vol. I.

SCARLET FEVER.

The period of incubation, while uncertain and variable, averages seven or eight days. At its expiration the symptoms marking the onset of the disease come on suddenly, with very little prodromal malaise or other evidences of infection. Vomiting, headache, rapid rise in temperature, hot, dry itchy skin and unusual increase in the pulse-rate, with some complaint of soreness of the throat, will all be noted inside of twelve hours, and at this time, too, the rash may be fairly well developed. Commencing on the chest as a diffuse erythematous blush with pin-point sized spots of more distinctly reddish tinge dotting the skin here and there, the eruption spreads to the neck and face and downward over the lower half of the body. In severe cases, often termed malignant, the evidences of septicæmia are marked in the pronounced depression, delirium, muscular twitching, carphologia, involuntary bowel movements, and at times in a non-appearance of the rash, or its recession after having once shown itself. The diagnosis is made, therefore, upon the knowledge of exposure about a week before, the sudden onset of the disease, with vomiting, sore-throat, high temperature, rapid pulse and the characteristic rash.

FIG. 2.



Temperature in scarlet fever.

The **temperature** in typical cases is above normal for about ten days, *i. e.*, until after the rash has disappeared. For the first three or four days it will range between 102° F. and 105° F., gradually disappearing. Mild cases occur where at no time does the temperature exceed 102°. A careful observation with the thermometer will often show that a child, apparently with no fever, after the eruption has been, perhaps, for three or four days invisible, will have an evening rise to 99.5° or 100°, an evidence that the disease process has not entirely ceased and a warning to the physician against a too early relaxation of the restraints placed upon these patients in the hopes of avoiding some of the later unfortunate complications.

The **pulse** is unusually rapid and is slow in returning to its normal beat. A pulse of over 140 in a child of ten years is more common than one of 120, and this, even though there may be no hyperpyrexia. A lagging and intermittent pulse is at times noticed during convalescence, and especially where a renal complication occurs.

The **rash** is generally diffused over the entire body, of a bright scarlet color, which fades on pressure, quickly to return, and looking as though thickly peppered with spots of a more pronounced and darker scarlet; these spots are often very slightly elevated above the surface of the skin. In a very peculiar manner the rash spares the skin about the lips and the alæ nasi, the face with its scarlet blush, and the white area about the mouth, presenting a picture so characteristic that upon its appearance alone a diagnosis can at times be based. In some cases the eruption is only plainly marked at the flexures of the joints or over the parts that have been subjected to pressure by contact with the bed, as over the scapulæ and the buttocks. Minute vesicles, sudamina apparently, are at times seen. In malignant cases a purplish hue has been noted or a complete failure of the rash to appear. Some patients go, through an attack without any rash.

The **tongue** is coated and soon shows prominent red papillæ giving it the strawberry appearance, more marked as the coating clears off, as it does after a few days, leaving the tongue fiery red. In severe cases the tongue is dry, lips parched, and teeth and gums covered with sordes.

The **throat** is early uniformly reddened to a dark scarlet hue; the tonsils are enlarged and in many cases covered with a dirty, grayish deposit, which, were it not for its failure to be strongly attached to the underlying structure, and its lack of the firmness of the diphtheritic membrane, might very easily be confounded with a genuine diphtheritic deposit. Bacteriological examination will fail to reveal the Klebs-Lœffler bacillus of diphtheria unless a secondary infection have occurred, which undoubtedly happens at times. The scarlatinal sore-throat is usually very painful; an ulcerating surface is often seen on the tonsil after the grayish film has disappeared, and the glands at the angles of the jaw are enlarged.

Soon after the rash has faded, usually at the expiration of ten or fourteen days, the outer layers of epidermis peel off in strips. This can be noted first about the nails. The thickened epidermis of the hands and feet is the last to be shed.

Variations from the typical cases are constantly met with. The malignant cases have already been referred to. Light cases are often difficult or impossible to diagnose. Cases are encountered where the only symptoms are severe sore-throat, temperature never above 102°, and no sign of a rash. Yet desquamation, the later occurrence of the disease in other members of the family or the advent of acute nephritis, fully establish the diagnosis. In one family the initial case was one of scarlatinal angina, but no rash. Inside of ten days two children were dead from a most malignant form of the disease, while a fourth child merely had a little stiffness of the neck, a slight enlargement of the glands, and for forty-eight hours a temperature of 99° to 101°.

Complications. **Middle-ear inflammation** is common. An easy channel of infection of the middle ear is afforded by the Eustachian tube, and from the inflamed throat we may have in succession an inflamed Eustachian tube, inflamed middle ear, often suppurating, and inflamed mastoid cells. Suppurative mastoiditis is a complication to be dreaded, as there is danger of a septic sinus-phlebitis with its attendant danger of meningitis, encephalitis, or pyæmia. Careful disinfection of the throat and posterior nares is in a measure instrumental in avoiding these unfortunate complications as well as the suppurative inflammation of the glands of the neck, which occasionally is met with.

Scarlet fever has an unenviable notoriety, even among the laity, for causing **acute nephritis.** At a time when all active manifestations of the disease have given physicians and parents a sene of security, the puffy face, suppression of urine, or passage of only a small amount of high specific gravity, richly albumnous, containing blood, numerous tube-casts, leucocytes, and epithelium, the vomiting, headache, rise in temperature, perhaps stupor or convulsion—all these give unmistakable evidence of the acute renal inflammation with its serious uræmic manifestations. The safe way is to examine the urine of a scarlet fever patient at frequent intervals in light as well as in severe cases, and during the convalescence as well as during the fastigium of the disease. A trace of albumin during the febrile stage is not significant of a genuine inflammatory lesion of the kidney. Dropsy without albuminuria has been observed during convalescence, attributed by some to the co-existing anæmia and by others to renal disturbance, as evidenced by the casts found in the urine.

A form of **synovitis**, usually spoken of as **scarlatinal rheumatism**, may occur during the course of the disease. Pain, swelling, redness of the joints—and several are usually affected—with some elevation of temperature mark this complication. Infection with pyogenic microbes, and suppurative arthritis are rarely sequels.

Following scarlatina there is often a prolonged period of exhaustion, anæmia, and neurasthenia. Among the less common complications and sequelæ are endocarditis, pericarditis, pleuritis, optic neuritis, parotitis, epilepsy, chronic conjunctivitis.

CHICKEN-POX. VARICELLA.

Varicella or chicken-pox is often overlooked by parents, so slight are the constitutional disturbances and so insignificant the skin lesion. The prodromes, after an incubative period of about ten to fourteen days, are usually little more than lassitude, trifling headache, possibly a temperature of 101°. The child may be somewhat cross and peevish, and a little restless at night. The rash appears at the end of twenty-four or forty-eight hours, usually first upon the chest, in the form of minute papules, rapidly becoming transformed into vesicles with clear, watery contents, and with a very narrow hyperæmic areola surrounding them. When lacking the areola, as is the case at times, the skin may look, as Fagge says, as though sprinkled with drops of water. If unbroken by pressure or scratching, the contents, before the vesicle ruptures or shrivels, may become lactescent. After rupture a small, grayish-yellow scab is seen, often bloodstained and upon a reddened base—these effects largely the result of the child's picking and scratching. The number of vesicles varies from no more than a dozen scattered over various parts of the body to a number sufficient to leave but small areas of unaffected skin. Usually well developed inside of twenty-four hours, the vesicles may continue to appear in crops for two or three days. No part of the surface of the body is exempt. They are found on the scalp, on the penis, and often on the roof of the mouth. A slight pit is occasionally left when the crust falls off.

Occasionally exaggerated symptoms of varicella are seen, and even fatal results have been recorded. Complications and sequelæ are exceptional, though severe bronchial catarrh, cervical adenitis, and albuminuria have been noted during the course of the disease, and anæmia and acute nephritis as sequelæ.

VARIOLA. SMALLPOX.

At the expiration of ten to fourteen days from the time of exposure the patient has severe headache and backache, often a chill and vomiting. At the same time there are rapid rise in temperature and symptoms of profound depression. At the end of three days a papular eruption appears, becoming changed successively to a vesicle, pustule, crust, and scar. The duration of the disease is three weeks.

Before the appearance of the rash a diagnosis, except during the prevalence of an epidemic, cannot be made positively. Suspicion as to the true nature of the oncoming infectious disease should be aroused by the severity of the chill, by the excruciating pain in the back and head, and by the sudden rise of temperature to 104° or 105°, as well as by the great prostration—strong men stagger like drunkards under the shock of the attack. Vomiting may be pronounced for a few days, and convulsions in children may be substituted for the chill of adults.

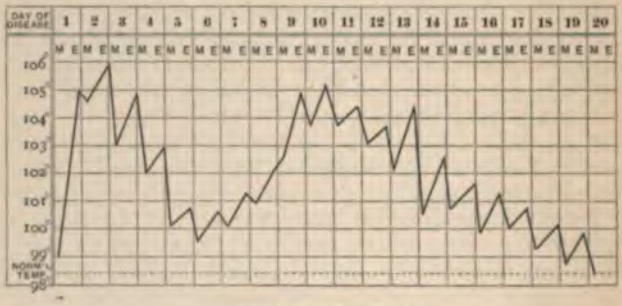
The rash. Preceding the appearance of papules an erythematous blush is often observed, especially in the axilla or on the inner surface of the thighs. A petechial eruption is less frequently noted over the lower abdomen and extending as low down as to the knees.

At the end of the third day from the onset papules of a shotty feel seem to lift themselves up from beneath the skin of the face and scalp, and at times of the wrists, and, extending from the face downward, they cover the entire body.

On the sixth day of the disease, *i. e.*, the third from the commencement of the eruption, the papule becomes transformed into a vesicle with a central depression or umbilication. The liquid contents are at first translucent and are contained within the meshes of the reticulated structure of the vesicle. Gradually the cellular elements in the liquid increase in number, the fluid becomes grayish and yellowish, and the vesicle is, in fact, transformed into a pustule, oftentimes with a bulging, rounded contour, the umbilication having disappeared, and surrounded by a swollen, reddened, brawny areola. By this time many of the lesions may have coalesced, constituting the *confluent* variety of smallpox. By rupture of pustules, or by their shrivelling, crusts are formed which, at the end of five or six days, drop off, leaving, where the inflammatory destruction has invaded the true skin, a pigmented depression or scar which slowly whitens out, leaving the characteristic smallpox pit formerly so often seen. The thick, outer layers of the skin on the hands and feet may be shed in patches, as in scarlatina. During the pustular stage the deformity of the features may be extreme, the swollen face with closed eyelids and tumid lips, the pustules, many of them dripping pus, the crusts, making such disfigurement that friends may well fail to recognize acquaintances. The odor from the patient is oftentimes extremely offensive. Vesicles may be present on the mucous membranes, as of the mouth, œsophagus, larynx, eye, or nose.

The **fever** remains for four days high. Then, as the papules are fully out, there is a marked drop in temperature. As suppuration begins on the ninth day, the secondary fever sets in, often reaching as high a point as the initial fever, 105° or 106°. It disappears with the drying of the pustules and the shedding of the crust, usually being normal by the twenty-first day.

FIG. 3.



Temperature in smallpox.

During the suppurative fever complications are liable to occur, such as laryngitis, pneumonia, bronchitis, endocarditis, keratitis, panophthalmitis.

Many of the deaths from variola take place during this period, the septic intoxication being more than the system can withstand.

Varieties. **Discrete** where pustules do not coalesce; **conflu-**

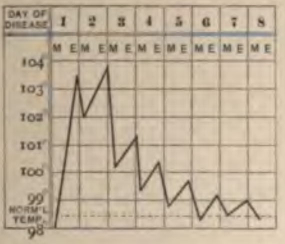
ent where two or more pustules become merged into one. Usually a more severe form of the disease than the discrete. Confluence most marked on the face.

Hemorrhagic. Where, even before the development of papules, subcutaneous or submucous hemorrhages occur. Bleeding from the nose, lung, stomach, or bowel may occur. Hæmaturia is frequently noticed. Almost uniformly fatal before the sixth day and marked even during the invasive stage by symptoms of profoundest depression. In a second variety of hemorrhagic smallpox the hemorrhagic tendency is not manifest until pustulation begins. This likewise is a most grave form of the disease.

Abortive variola, where initial symptoms, with perhaps erythema, occur during an epidemic, but no distinct variolous eruption. This form of variola confers immunity against a second attack.

Varioloid. A form of variola modified by a previous attack or by vaccination. The initial symptoms may be very similar to those of an ordinary smallpox. But the rash appears on the second day, or early on the third, and rarely goes on to suppuration or the formation of scars. The papules appear irregularly scattered over various parts of the body, sometimes few in number, sometimes many. They do not always appear first upon the face. There is no suppurative fever. The disease is a true smallpox, running an irregular, rapid, and abbreviated course. Its diagnosis is therefore more difficult than that of unmodified variola, though none the less important because the milder form of the disease may be the origin of the more serious form in others.

Fig. 4.



Temperature in varioloid.

Measles.	Scarlet fever.	Smallpox.
Period of incubation ten to fourteen days.	Variable, but averages seven days.	Ten to fourteen days.
Prodromal coryza, bronchial cough, and fever 99° to 102° for three to four days.	Prodromes of brief duration. Before rash, vomiting, sore-throat, rapid pulse, temperature 104° or 105°.	For three days preceding rash high temperature, 104° to 106°, severe headache and backache, great depression; ushered in by chill or convulsion.
Rash, papular, running into blotches.	Rash scarlet, erythematous blush with redder prominent spots.	Rash papular, then umbilicated vesicular, later a pustule, a crust, a scar.
Begins on face, and is most marked there.	Begins on chest and is most marked there.	Begins on face, and is most marked there.
Lasts about four days.	Lasts five or six days.	Lasts from papule to desiccation about two weeks.
Desquamation in branny scales.	Desquamation in strips.	No desquamation save dropping off of crusts.
Temperature 103° to 104°, till rash well out, then drops by crisis. From beginning of prodromal fever to total disappearance about one week. No secondary fever.	Temperature 104° to 106° for about four days. Recedes by lysis, totally disappearing by end of second week. Pulse very rapid.	Temperature 104° to 107° for three days, then much lower for about five days, rising again on ninth day to 104° to 106°. Disappears by lysis. Lasts altogether two to three weeks.
Tongue coated; perhaps red at edge.	Strawberry tongue.	Tongue coated, often swollen and painful; may be salivation.
Rarely cerebral symptoms.	Cerebral often present.	Cerebral symptoms and profound depression may be grave.
Coryza and bronchitis.	Sore-throat and enlarged glands.	Sore-throat and hacking cough may occur True bronchitis a complication.
When rash is *fully* out symptoms abate.	No amelioration of symptoms with appearance of eruption.	When rash *begins* to develop relief from pains, fever, and vomiting
Respiratory complications and sequelæ commonest.	Kidney, joint, middle ear, and serous membrane inflammations commonest.	Complications and sequelæ, considering the gravity of the disease, rare. Pneumonia, eye inflammations, diarrhœa, adenitis of occasional occurrence.

TYPHOID FEVER.

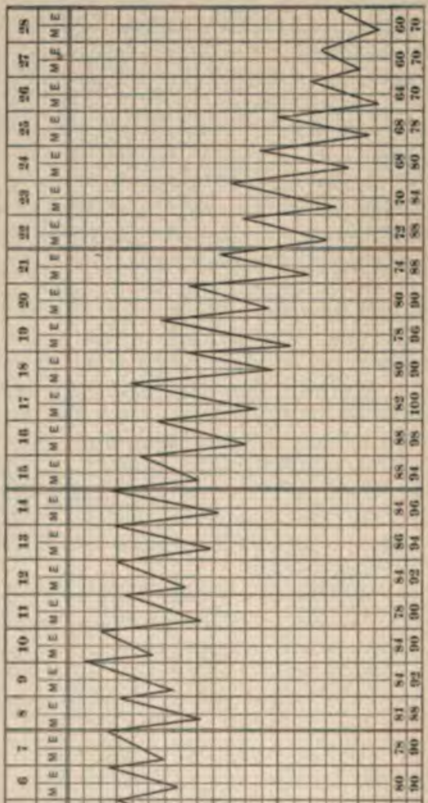
Typhoid fever is oftenest met with in young adults and especially in our large cities, among the newcomers. It is most prevalent in late summer and autumn.

The initial symptoms of typhoid fever come on as a rule gradually and insidiously. For several days there are lassitude, anorexia, headache. Chilly sensations may then appear, with a feverish flush toward night. Epistaxis is common. By this time the patient feels so ill as to have given up work, and he consults a physician. The fever at the end of a week has reached its height, the patient has steadily grown weaker, is dull and apathetic, perhaps delirious at night; he has an enlarged spleen, a tympanitic abdomen upon which a crop of rose-red spots may make its appearance, and has perhaps developed a diarrhœa. For ten or twelve days he continues in this state, growing emaciated, the nervous depression shown by the clouded mind, the muscular twitchings, perhaps by a feeble dicrotic pulse, the temperature remaining day after day at 102° to 105°. If complications do not arise and the strength of the patient be sufficient to withstand the toxine intoxication, the self-limiting character of the disease is now manifested by a gradual disappearance of fever with coincident improvement in other respects. The annoying bronchial cough abates, the tympany vanishes, the mind grows clearer, the pulse stronger, the dry, coated tongue clears off and grows moist, the appetite returns, and convalescence is fully established.

It is impossible to depict a typical case of typhoid, for no two cases are alike. Among the prominent features of the disease demanding special attention are the following:

The Fever. The course of the temperature can be best described by dividing it—somewhat arbitrarily, to be sure—into four stages corresponding to the four weeks of the disease. The first week is the initial period or the *stadium incrementi.* During this week the fever rises gradually from one to two degrees a day, until by the fifth to the seventh day it has reached 104° to 105.5°. It is noted here, as throughout the course of the disease, that a morning remission is almost constantly present. Baruch believes that where a rectal temperature of 102.5° in the morning and 103° in the evening has persisted for three successive days, and where the temperature is not reduced by a bath of 75°, with friction, the diagnosis of typhoid is established. The resistance of the rectal temperature to a bath of 75° for fifteen minutes, with friction, is an almost certain test of typhoid fever. The details of the method of bathing are in the *New York Medical Journal*, September 2, 1893. During the second week, or the *stadium*

FIG. 5.



Temperature in unmodified typhoid fever.

fastigii, the temperature remains constantly high in the evening, varying from 103° to 105°, the morning drop being about one to two degrees.

The third week, or *stadium ambiguum*, still shows the temperature high in the evening, but morning remissions are more marked, and there is an irregularity about the temperature very different from its behavior the week before. A temperature of 100° in the morning, for instance, may be changed by evening to 104.5°. Antipyretics and antipyretic measures, as bathing, will be found in this period to exert a more powerful influence much more readily than during the preceding week. The dose of phenacetin that on the ninth day barely brought the temperature of 104° down to 102° will now bring it down to 99° or even cause a drop to subnormal, with accompanying cyanosis and signs of collapse. The fourth week, or *stadium decrementi*, shows the gradual decline of the fever, the temperature often being normal for several mornings, while a slight evening rise still persists. Complete apyrexia is usually obtained by the twenty-eighth day.

Modifications of this febrile course are frequently observed. By the fourteenth or fifteenth day the lysis may begin and be completed by the end of the third week. In other cases, where, too, no complication can be discovered, the fever drags on through the fifth or sixth week, with a somewhat erratic course, though all the time with a tendency to the remittent type. And there can be no question that certain cases, very rarely seen, are entirely lacking in elevation of temperature—apyretic typhoid.

Pulse. A slow pulse, despite high temperature, is so characteristic of typhoid that its value from a diagnostic and prognostic point of view should not be overlooked. In a bed patient—*i. e.*, one whose heart-beat is not accelerated by previous walking, as to the office, or other exertion—a pulse of 80 or 90, with a temperature of 103° or 104° should always lead to the suspicion of typhoid. On the day that I write these lines I have seen a girl with typhoid whose temperature was 105.4°, pulse 100. In women the rate averages higher. The first few days out of bed during convalescence often cause a pulse (that during three weeks in bed when the thermometer has registered from 102° to 105°, has never exceeded 100 beats to the minute) to jump to 130 or 140. In other cases bradycardia is observed. In a male a pulse of over 104 should be looked upon as one needing the most careful watching. Dicrotism is often noticed during typhoid. Occasionally the pulse becomes intermittent. In cases severely ill, and where asthenia is daily becoming more pronounced, the pulse has a tendency to become more rapid, and often very insidiously increasing a few beats every day. Such a pulse needs careful guarding, for fear of its sudden giving out.

Digestive System. The tongue in many cases is moist throughout, usually coated, and often flabby. In the severer forms, those that might be termed malignant, it soon becomes dry, covered with sordes, as are the teeth and lips, and perhaps cracked and bleeding. As convalescence is about to begin the edges become moist, the coating gradually clears off, and the tongue assumes the normal appearance. Pharyngeal ulcers with a grayish-white coat are occasionally seen. Acute gastric catarrh will occasionally cause vomiting at the commencement of the disease. Vomiting during the course is rarer, and at times may be traced to the abundance of drugs forced down the patient.

Constipation, often present during the first week, will usually give place to diarrhœa in the second week. Liquid stools of peculiar odor and pea-soup appearance are sometimes spoken of as typhoid stools. In the semi-stupid state of the typhoid the stools may be passed involuntarily. Constipation may be present throughout the course of the disease. Tympanites is present in the majority of cases. At times it is extreme, and by pressure a source of discomfort and danger. There are sometimes borborygmi, and gurgling can generally be elicited in the right iliac fossa by pressure. Some tenderness is usually found here.

Respiratory System. A bronchitis is so commonly present as to be looked upon as one of the accompaniments rather than complications of typhoid. A hacking cough, a few moist and dry râles give evidence of the bronchial catarrh. At times it is extreme, and especially where patients have lain on the back for a long period of time does it become marked in the congested areas posteriorly. Laryngitis and laryngeal ulcers are occasionally found.

Genito-urinary System. A trace of albumin is frequent. Retention of urine may occur in patients apparently not very ill. Incontinence of urine and involuntary urination are found in some of the sickest patients. Ehrlich's diazo-reaction is usually to be made out during the height of the disease.

Ehrlich's Reaction. Two solutions kept in separate bottles are necessary. One contains a ½ per cent. solution of hydrochloric acid saturated with sulphanilic acid; the other a ½ per cent. solution of sodium nitrite. Forty parts of sulphanilic acid solution are added to one part of the sodium nitrite solution, and the two thoroughly mixed. Of this mixture 1 c.cm. is added to 1 c.cm. of urine, and the two shaken, a yellow solution resulting. One c.cm. of ammonium hydrate is allowed to trickle down the side of the test-tube. A garnet ring between the yellow solution beneath and the ammonia above constitutes the diazo reaction, regarded by Ehrlich as, with few exceptions, peculiar to typhoid. A light-brown or yellowish ring is not abnormal.

Dr. A. R. Edwards examined the urine of 130 cases of typhoid in the Cook County Hospital, and obtained the reaction in all save two.[1] Its absence, therefore, may be regarded as presumptive evidence against typhoid, though not by any means positive. The detection of the reaction is of very trifling value, however, as it has been found by many observers in enteritis, malaria, tuberculosis, etc. It is common in measles. Jaksch regards it as merely an unreliable test for acetone in the urine.[2]

Skin. A typhoid rarely sweats during the height of the disease, save as the result of antipyretics. Between the sixth and fourteenth days slightly elevated papules, rose-red in color, seldom as large as a split pea, appear on the abdomen and trunk, and rarely on the extremities. The color disappears on pressure. The number of spots varies from one or two to several hundred. The spots may appear in crops. Desquamation is not marked. Post-febrile alopecia of the scalp is common, the hair usually being replaced by a new growth.

Nervous System. Mental dulness and apathy are characteristic of typhoid even quite early. A typhoid takes little interest in his surroundings, and usually, even when at his worst, will express himself as feeling "first-rate" or "better." Abdominal pain is usually slight, unless complications exist. Muttering delirium and more rarely violent delirium are observed at times. Subsultus tendinum, tremulousness of the tongue, retention of urine, show the altered condition of the nervous system. Headache, one of the early evidences of the disease, is seldom complained of after the first week. Paralysis of separate muscles or groups of muscles, hyperæsthesia of the skin, hysteria, mental weakness, melancholia or other psychoses may be phenomena of convalescence.

Complications and Exaggeration of Symptoms. Stomatitis in its various forms may occur, especially in those greatly debilitated by the disease, and to the cleanliness of whose mouths but little attention has been given. The catarrhal œsophagitis and *gastritis* that accompany all the acute infectious diseases may be of an aggravated form, especially early in the history of typhoid, as shown by pain on swallowing, nausea, vomiting, and distress after eating. **Diarrhœa,** so often present, may become so prominent a feature as to be regarded in the light of a complication. It is severest where the colon is involved in catarrhal or ulcerative inflammation. Distention of the paretic intestines is productive of *tympanites,* which, when excessive, is an element of danger. It is important to discriminate between this form of tympany and that of peritonitis, with the tenderness on pressure, wiry pulse, pinched features, etc. **Hemorrhage** from the bowels rarely is met with earlier than the second week. When slight in amount it can only be recognized by the appearance of blood in the stools, bright red if recent, dark if having remained in the bowel for any length of time. Larger losses of blood give evidences of acute anæmia, perhaps even before the stools show any trace of blood. The sense of weakness, yawning, pallor of face and lips, anxiety, rapid fluttering pulse, sudden drop in temperature, are as much the cause of alarm when occurring in a typhoid as in the woman recently delivered. Hemorrhage occurs oftenest in those who have suffered from diarrhœa and are regarded as the sickest. **Perforation,** on the contrary, is as liable to occur in the light as in the severe case. It is most commonly met with in the third and fourth weeks. If the patient's nerves be not too dulled and blunted at the time of perforation, he will usually cry out with a severe, sudden pain in the lower zone of the abdomen, and the evidences of shock are at once manifested. With the onset of peritonitis, reaction may occur, the pulse becoming quick, wiry, and rapid, and the temperature rising. Tympany, abdominal tenderness, pinched features are as in septic peritonitis from any other cause. Signs of fluid and gas in the abdominal cavity can perhaps be made out. Other cases are much more insidious, but little pain being complained of. It is probable that many typhoids, said to have died of asthenia or of sudden collapse, have been the victims of perforation peritonitis with an insidious onset. Very rarely peritonitis is met with where autopsy fails to show perforation.

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[1] Medical News, April 2, 1892.
[2] Prag. med. Wochenschr., 1891.

Acute splenic tumor is so marked in typhoid as to be always sought for. Percussion will yield increased area of dulness. Palpation is often rewarded by the detection of the smooth edge of the spleen creeping out from beneath the left costal arch.

Palpation of the spleen should be made with the patient on the back, the examiner to his right, the finger-tips pressed upward under the ribs, the hand lying flat upon the abdomen, the patient breathing quietly. Practice is of much value in palpation of the spleen. Tympanites interferes greatly with its successful performance. Excessive stretching of the splenic capsule causes pain in the left hypochondrium. This will be complained of by perhaps one per cent. of typhoids.

Respiratory. *Epistaxis*, a common early symptom, may by its persistence or severity, become a cause of death unless checked by proper measures, such as plugging of the nares.

A slight *bronchial catarrh* and consequent cough cannot be looked upon as a complication. It is a part of the pathological changes incident to the disease. The dorsal decubitus, the inspiration of air passing, before its entrance into the bronchi and alaeoli, through a foul nose, mouth, and pharynx, and carrying in various micro-organisms, the morbidly inactive nerve-endings, which fail to respond to even an unusual stimulus by a cough, all this favors the development of a severe grade of bronchitis, involving perhaps the capillary tubes and leading to localized patches of pneumonia. Inhalation pneumonia or genuine croupous pneumonia may be a serious and fatal complicating disease. It is of importance to remember that in a typhoid these respiratory complications may fail to show the classical subjective symptoms. The chill, pain, and cough, with characteristic expectorate, may be lacking, and the already high temperature may be but slightly increased by the complication. A careful daily watching of the respiration-record, with a physical exploration of the chest, is the only sure means of diagnosing these complications. The possibility of confusing a diffuse bronchitis of typhoid with an acute miliary tuberculosis is to be kept in mind.

A very rare complication is perichondritis, due, perhaps, to infection through laryngeal ulcers. This complication gives symptoms of œdema of the glottis, and usually demands tracheotomy. It is looked upon as a grave complication.

Without entering into a discussion of the question as to whether some of the suppurative processes found during or subsequent to typhoid may be excited by typhoid bacilli alone, or whether a mixed or secondary infection with the true pus microbes must be presupposed, suffice it to say that suppurative inflammation of the pleura, of joints, of bone, of the liver, of the parotid gland, and of mediastinal glands may occur.

Furuncles may be abundant. The diagnosis of each of these conditions is made in the same way as in cases of primary suppuration in these organs. Where careful cleanliness of the mouth is insisted upon parotitis is rare, as are furuncles where there is cleanliness of the skin. Suppurative mesenteric adenitis may account for some of the delayed convalescences where no demonstrable cause is found, as it does by the rupture of the pus sac, for an occasional suppurative peritonitis.

Pleurisy with a serous effusion is comparatively rare. Endocarditis of the so-called simple, or of the malignant, type is rare. Through extreme feebleness of circulation, and in other cases from undetermined cause, thrombosis, especially in the femoral vein, may occur. Pain, tenderness, and often induration, with œdema of the extremity and a rise in temperature, enable us to make the diagnosis. Detached bits of the thrombus very rarely cause embolic obstruction of some distant vessel, especially in the lung. The œdema from femoral thrombosis sometimes persists for months following recovery from the typhoid.

Cystitis is usually due to catheterization. Nephritis, giving a severe form of disease spoken of as nephro-typhoid, may occur.

Peripheral neuritis is not rarely met with. Post-febrile insanity, often of a melancholic or demented type, is rare. Feebleness of mind is commoner.

Erysipelas of the face usually causes death when complicating typhoid. A petechial eruption on the trunk, pelioma, is probably produced by body-lice. I have seen it once, and then the lice were present. Typhoid and pregnancy usually mean miscarriage.

A bed-sore during typhoid is a source of annoyance and danger, septic infection being more than once traced to this source. It is at times, as where intestinal hemorrhage occurs, and absolute rest is insisted upon, unavoidable, and yet one feels inclined to agree with Henry when he says: "A bed-sore has no legitimate place in the clinical history of typhoid fever. It is neither a complication nor an intercurrent affection; it is a blunder."[1]

Varieties. **Abortive typhoid** is a name given to cases where all the premonitory and initial symptoms give evidence of the onset of the disease, but after a week or ten days of fever the patient is apparently fully recovered. Some authors deny that such cases are true typhoid.

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[1] Hare's System of Practical Therapeutics, vol. ii. p. 315.

Walking typhoid, where the patient does not go to bed for days after the commencement of the disease, or not at all during its course. Hemorrhage from the bowel or perforation has in some instances been the first evidence to the patient that he was in need of a physician's advice.

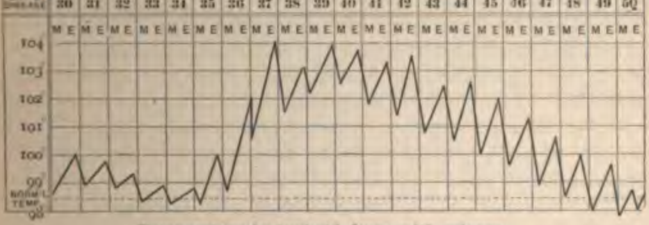
Apyretic typhoid, where there is no elevation of temperature during the progress of the disease, but the other symptoms, or autopsy, prove it typhoid.

Of three cases diagnosed apyretic typhoid in the Cook County Hospital two recovered; the third died, the autopsy showing typical typhoid lesions. Diagnosis in such cases has to be made partly by exclusion and by the positive signs of weakness, hebetude, eruption, tympany, splenic enlargement, and Ehrlich's reaction in urine.

In **children** high temperature with correspondingly slight depression of bodily strength is characteristic. A rapid course —often only fourteen days, few intestinal complications, and usually favorable outcome are common in children. In the **aged** greater prostration and a more insidious onset and progress mark the disease, and the mortality is high.

Relapse and Recrudescence. A relapse is to be looked upon as a new infection with typhoid bacilli. After a period of quiescence and apyrexia the temperature rises, as at the beginning, though more rapidly; new spots appear, the spleen again enlarges, and we have, in short, a repetition of the original attack. Its duration, is, however, briefer.

FIG. 6.



Temperature in typhoid fever with relapse.

By recrudescence is meant a temporary rise such as is met with often during convalescence, from nervous excitement or from dietetic error.

Differential Diagnosis. But a few of the diseases from which typhoid has to be distinguished can be mentioned. For details the chapters on these diseases may be consulted. It is, like syphilis, a great imitator of disease, or perhaps it should be said that many other diseases imitate typhoid.

In doubtful **malarial cases**, where there is absence of the characteristic periodical fever and chills, of herpes, etc., the detection of the plasmodium malariæ in the blood furnishes the sole positive means of diagnosis. Typhoid and malaria may coexist.

Cases are sometimes seen with such marked chest symptoms as to lead to a suspicion of **diffuse tuberculosis**. Only by the positive signs of typhoid and a careful balancing of all the evidence can a sure diagnosis be arrived at here. In some cases it must remain doubtful. Blood examination may show tubercle bacilli.

Suppurative processes lead to mistakes oftener than any other condition. Among these may be instanced empyema, abscess of the liver, appendicitis, osteomyelitis, septicæmia, ulcerative endocarditis. The most careful examination for local evidences of inflammation, and at times the careful observation of the patient for a few days, are necessary to avoid error in such cases.

Among other diseases that must be excluded may be mentioned cerebro-spinal meningitis, gastro-enteritis, influenza, trichinosis, typhus fever, and some forms of uræmia, acute miliary tuberculosis.

The error in diagnosis seldom consists in calling typhoid fever appendicitis, osteomyelitis, trichinosis, miliary tuberculosis, empyema, hepatic abscess, but the opposite. Without some positive evidence of typhoid—*e. g.*, unmistakable rose-spots—the physician must ever be on the alert for the signs of other disease, and must day after day make careful examination of all organs as the possible sites of septic inflammatory processes. The positive diagnosis has oftentimes to be withheld for several days, or in some cases, unfortunately—*e. g.*, at times in miliary tuberculosis, malignant endocarditis, cryptogenetic septicæmia—reserved for the post-mortem revelations.

Bacteriological examination of blood drawn from the spleen is scarcely to be commended as practical, nor is the examination of the stools for the bacillus typhosus of any value save at the hands of an expert bacteriologist.

To recapitulate, the most valuable points in favor of a diagnosis of typhoid fever are:

1. Gradual onset with headache, malaise, anorexia, epistaxis, chilliness.

2. Gradual rise of temperature with distinct morning remissions; pulse slow.
3. By the end of first week bronchitis, mental apathy, rose-spots, more or less tympany, splenic tumor, often diarrhœa.
4. Absence of signs of local inflammatory or suppurative processes—*e. g.*, endocarditis, encephalitis, hepatitis, pleuritis, etc.
5. Ehrlich's reaction in the urine, Baruch's bath-test may confirm diagnosis.
6. Examination of blood in uncomplicated cases shows no leucocytosis. With suppuration, leucocytosis is usually marked.

TYPHUS FEVER.

Typhus fever is a disease rarely met with save among patients with poor hygienic surroundings, as where people are crowded together in tenement-houses, in jails or camps, or in ships.

The disease is highly contagious. The period of incubation is about ten or twelve days. Symptoms usually come on with little or no warning. A severe chill, pain in the head and loins, great prostration, vomiting, rapid pulse, and sudden rise in temperature mark the onset.

The **temperature**, rising rapidly to 103° or 104°, will have reached its height, 104° to 106°, by the fourth or fifth day. Slight morning remissions occur, and if the patient survive till the end of the second or beginning of the third week, defervescence by crisis terminates the febrile career. The pulse, rarely dicrotic, keeps pace with the fever, being usually full and bounding at the beginning, to become weak and more rapid as debility becomes more marked.

The virulence of the septic intoxication is early shown in the **nervous phenomena.** Delirium, muttering or more rarely maniacal, stupor, coma-vigil, subsultus all give evidence of the profound impression made upon the nerve-centres. The dull, apathetic look of the patient is the same as in severe typhoid, but is noticeable much earlier. The countenance is usually flushed, the tongue dry and foul, and from the fifth day on a rash covers the body. This is macular, of a dark-reddish color, often petechial, the individual spots usually larger than the rose-spots of typhoid. The peculiar appearance presented by the exanthem has given typhus its name of spotted fever. The rash is at times papular before becoming petechial. It spreads from the trunk to the extremities and face.

There are many variations from the typical course of the disease, mild cases with symptoms minimized, and malignant cases with exaggerated manifestations and early death, being found during epidemics. Sporadic cases are difficult to distinguish from typhoid fever.

The differential diagnosis is to be made where possible by considering that in typhus the onset is more sudden, marked by a chill, profound depression, nervous symptoms, and high temperature earlier developed. There is a deeper flush of the face in typhus. Tympanites is rare, constipation the rule. The eruption is darker, more generalized, and much more frequently petechial. Death or recovery occurs earlier in typhus, the latter often preceded by critical drop of temperature, in marked contrast to the gradual defervescence of typhoid. No typhoid bacilli are detectable in stools or blood. In general it may be said that typhoid is insidious in its onset, slow in its progress, and tardy in retiring, while typhus is frank and open in attack, more rapid in its advance, and of quick retreat.

EPIDEMIC PAROTITIS.

Mumps is a disease characterized by a non-suppurative inflammation of the parotid gland. It is contagious. Two or three weeks after exposure there is pain just in front of and below the ear, where a swelling is soon manifest. Movements of the jaw, as in masticating or talking, are restricted on account of the pain. In sixty hours the swelling has usually reached its height. In front of and below the ear, and sometimes beneath the ramus of the jaw, is a rounded swelling, not very hard, tender, and at times slightly reddened. The gland on the opposite side is usually involved by the third day. Resolution is complete in a week or ten days.

The constitutional disturbance in many cases is slight—malaise, anorexia, temperature 101°. In other cases the temperature may reach 103°–105°; there are severe headache, vomiting, great weakness, and even a typhoid condition. Delirium in such cases is not uncommon, and may be due to an accompanying meningitis, a complication responsible for some of the rare fatal cases. Among other complications may be noted the not infrequent orchitis in males or the rarer inflammations of the breast or ovaries in the female. These complications are prone to appear during convalescence. Deafness sometimes follows mumps. Disorders of digestion lasting several weeks occasionally follow mumps.

There is rarely any difficulty in diagnosis, though it must always be remembered that an inflammation of the parotid gland, usually suppurative, may occur as a complication of some other disease, as scarlatina or typhoid fever, where infection probably takes place from the mouth by way of Steno's duct.

The disease must also be differentiated from the diffuse and brawny swelling of the neck that sometimes accompanies diphtheria and scarlet fever. Examination of the throat, the presence of a rash, etc., enable the diagnosis to be made in these cases.

In rheumatism of the temporo-maxillary articulation there are usually the coincident swelling and pain in other joints, the acid urine, copious sweats, etc.

PERTUSSIS.

This contagious disease is oftenest seen in children below the age of ten. It is readily diagnosed when fully developed by the severe cough occurring in paroxysms, accompanied by the congestion, even to cyanosis of the face, often by vomiting or the raising of thick glairy mucus and by the deep inspiration which occurs at the end of several violent expiratory efforts, and which is accompanied by the characteristic whoop. In infants the whoop may be lacking. At the beginning the symptoms are those of an ordinary cold—reddened eyelids, acute catarrh of the nose, bronchial cough, with perhaps slight temperature. In a week or ten days the convulsive nature of the disease becomes manifest. The number of paroxysms varies from five or six to thirty or forty in twenty-four hours. The disease usually lasts from three to eight weeks, the symptoms gradually disappearing.

The mortality rate is high in young and weakly children, both because of the somewhat frequent occurrence of capillary bronchitis and broncho-pneumonia as complications, and because of the asthenia caused by loss of sleep and loss of nourishment. The frequent vomiting prevents absorption of food that is taken into the stomach. Pulmonary tuberculosis is seen as a sequel at times. The enlarged bronchial glands are often slow in regaining their normal size. The violent coughing-fits may cause hemorrhage, not alone beneath the conjunctiva, from the nose and pharynx, but even from the meningeal bloodvessels.

The sharp lower incisors may cause a laceration of the mucous membrane during the convulsive cough, so that a little ulcer may be formed under the tongue close to or involving the frænum. Measles may precede or follow pertussis.

INFLUENZA.

Influenza, grippe, or epidemic catarrhal fever, as it is sometimes termed, attacks during its prevalence people of all ages and conditions, proving fatal by virtue of complications in a large percentage of the aged, infants, and the feeble and those already afflicted with some other disease, as tuberculosis, diabetes, chronic nephritis.

For purposes of description, a respiratory form, an intestinal form, and a nervous form are spoken of as distinct, though in practice these forms are often, if not usually, combined.

In the first form the mucous membranes of the respiratory tract are most markedly affected. The symptoms of an ordinary cold are first noted—coryza, sneezing, cough; but the pains in the head, chest, back, and muscles, and the prostration as well as the fever, which may rapidly reach 103°–105°, mark the disease as something severer than an ordinary bronchial catarrh. The fever, aches, and pains may disappear in twenty-four hours, or only at the end of a week, but the exhaustion, and oftentimes the irritating, dry, cough continue for days, or even weeks.

In other cases, the *intestinal* cases, vomiting and diarrhœa are pronounced features. Frequent watery, painless, offensive stools are passed, the prostration being marked, though the fever may not be very high. The resemblance to typhoid fever for two or three days may be marked.

In the *nervous* form of the disease headache is severe, neuralgias of separate nerves can be made out, delirium may be an early symptom, and neurasthenia, neuritis, depression of spirits, or even insanity, one of the unfortunate sequelæ. In its early manifestations some cases, were it not for the initial coryza and bronchitis, might, from the vomiting, delirium, and fever, be mistaken for meningitis.

During the epidemics in Chicago in 1890, '91, and '92 the disease was truly protean in its manifestations, some patients prostrated for days and in a typhoid state; others, especially children, taken suddenly with headache, delirium, temperature 105°, rapid pulse, and in twenty-four hours with normal temperature and pulse and no complaint of pain. Many patients were not ill enough to give up work or even to consult a physician. In 1892 the enteric type prevailed, in some instances the manifestations being almost dysenteric.

The complications of influenza are numerous. The most common, and, at the same time, the most serious, are capillary bronchitis and pneumonia. Pleurisy, often suppurative, is not infrequent. Functional cardiac disturbances, endocardial and periodical inflammations, various nervous disorders, more particularly pronounced nervous prostration or neurasthenia, are among the many complications and sequelæ frequently noted.

The diagnosis during an epidemic is usually easy, as the physician is on the lookout for the disease. The early cases during an epidemic, and the stray cases lingering after, are more easily overlooked.

The main features to be relied on are the extensive involvements of the mucous membrane, the eccentricities of the temperature, the great nervous prostration, and the severe pains. Some patients who had experienced both dengue and small-pox declared that the headache and lumbar pains of grippe were far more torturing than in either one of the other diseases. In cases coming on with a sudden development of typhoid symptoms and consolidation of the lung it is difficult or impossible to discriminate between a primary croupous pneumonia and a pneumonia secondary to the influenza. In some cases too, pain in the chest, high fever, and the physical signs of congestion of the lung may mislead one to diagnose a pneumonia. Here one must await the development of twenty-four hours.

The discovery by Pfeiffer in 1892 of a bacillus in the sputum and blood of influenza patients, with the successful inoculation-experiments on apes and rabbits, with the confirmatory observation of Kitasato, Canon,[1] Cornil, Babes, and others, may make the diagnosis in doubtful cases more positive by examination of sputum and blood for the parasite. To examine the sputum Pfeiffer directs that a thin layer on the cover-glass, air-dried, should be gently heated and then stained for ten minutes in a 10 per cent. Ziehl's carbol-fuchsin solution. The poles of the small bacillus stain most intensely. The bacillus can be grown on sugar-agar (1½), showing in minute colonies, looking like drops of water.

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[1] Pfeiffer, Kitasato, Canon: Deutsche med. Woch., 1892, No. 2. Pfeiffer und Beck: Ibid., No. 21.

DENGUE.

This disease, confined to warm climates, is ushered in with chilliness, increased pulse and respiration-rate, pain in the head, back, and muscles, and, at times, with delirium. The temperature rises even on the first day to 103° to 106°. The joints are swollen, stiff, and painful, as in acute articular rheumatism. The lymphatic glands may be enlarged and tender. After two or three days a period of apyrexia and freedom from pain is reached, when the patient, were it not for the great prostration and weakness, would be regarded as well. An erythematous rash resembling that of scarlet fever makes its appearance a few days after the fever has disappeared, remains visible two or three days, and is followed by desquamation. More rarely the eruption consists of papules, wheals, or vesicles. With the appearance of the rash, or just as it is subsiding, a secondary fever sets in, with returning arthritic pains. After about three days recovery follows, though complete return to usual health and vigor is sometimes slow. Death and complications are rare. Organisms have been found in the blood by McLaughlin.[1] They may prove to be the causative agent of the disease and their detection would be an aid in diagnosis.

This disease has to be distinguished from rheumatism by the higher fever, rash, secondary fever; from scarlatina by the absence of sore-throat, late appearance of rash, and by the involvement of joints; from influenza by absence of respiratory symptoms, by the rash and secondary fever; from malaria by the joint-involvement, the rash, and absence of the plasmodium; and from yellow fever by the absence of icterus, frequent vomiting, hemorrhage, albuminuria, and by the secondary paroxysm.

RELAPSING FEVER.

This is a disease of extreme rarity in this country, and is chiefly met with in India, Germany, and other lands, among the filthy poor. Its cause is a spirillum, and the disease has been produced in man and in monkeys by inoculation. It is contagious, and epidemics have been extensive and serious. The period of incubation is from five to eight days.

The onset is usually unheralded by prodrome. The chill is severe, pains in the back and head are marked, as well as in the muscles, especially those of the calf of the leg. A rapid pulse, enlarged spleen, and high fever complete the picture of severe septic trouble.

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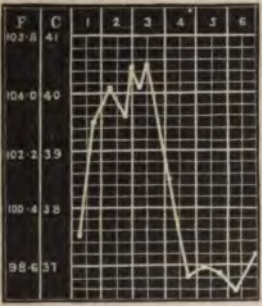
[1] Journal American Medical Association, June 19, 1886.

The course of the fever is characteristic. For from four to ten days, usually about six, the temperature ranges between 103° and 106°, there being slight remissions in the morning. Then an abrupt decline occurs, often at night, the temperature for a few hours being subnormal. Amelioration of symptoms is coincident with the drop in temperature. After a period of apyrexia lasting for a week or ten days a relapse occurs, the temperature and other symptoms being the same as before. Three or even five relapses have been noted, the rule being that each succeeding relapse is less severe than the preceding. Death is of rare occurrence. Complications are seldom met with, though iritis, laryngitis, pneumonia, acute nephritis, and suppuration in various organs are occasionally reported. An icteroid hue of the skin is of common occurrence.

FIG. 7.



Temperature in relapsing fever. (WUNDERLICH.)

The diagnosis, usually clearly made by the repetition of the high fever, is verified by an examination of the blood. A drop from the finger magnified enough to show plainly the red corpuscles (about 350 diameters) shows the slender, actively moving spirilla about five times as long as the diameter of the red corpuscles, to which they often communicate movements by which the attention is directed to the parasite. They are most abundant the day after the chill, disappearing as crisis approaches. Jaksch has noticed small rounded refractile bodies, resembling in appearance diplococci, which he is inclined to regard as the spores of the organism. The resemblance between malaria and relapsing fever both in clinical history and blood-findings is quite striking.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

This disease attacks by preference the young. It occurs sporadically at times, or prevails as a widespread epidemic, unhygienic conditions seeming to favor its development. The majority of cases occur in the winter or spring.

In the common acute form the first symptoms are chill or chilliness, often vomiting, extremely severe headache, and at times vertigo. There is great prostration. The muscles of the neck and back become rigidly contracted, causing the characteristic retraction of the head and bowing of the back, sometimes amounting to opisthotonos. Muscular spasms, tonic or clonic, may be marked, as in the arms and legs. Delirium is commonly present, and as the exudate increases stupor or coma may come on. The fever may be high, or even in severe cases may not exceed 102°. The pulse is in some cases unusually slow, sixty beats to the minute, notwithstanding the elevated temperature. Later it may become quite rapid. Irregularities in the behavior of pulse and fever are characteristic. Constipation is the rule. The urine may be normal or increased in amount. Occasionally albuminuria or glycosuria is reported. A petechial eruption, often in blotches, is seen in the greater number of cases, giving the disease the name among the laity of "spotted fever." Herpes labialis is as common as in pneumonia. Urticarial and erythematous rashes are at times seen.

As the disease progresses evidences of irritation or of pressure upon nerve-centres or nerve-trunks are manifest. Nystagmus, strabismus, ptosis, inequality of the pupils, blindness, deafness, partial facial paralysis, show the altered functions of the cranial nerves. Cutaneous hyperæsthesia is quite common. Muscular twitchings and more rarely paralyses may be noted. The cutaneous reflexes are usually preserved. The patellar reflex may be normal or diminished.

While the prognosis is grave, recovery may ensue, the symptoms gradually abating, and convalescence being prolonged over several weeks.

As in other epidemic diseases, all varieties as regards severity are noted, as the **malignant** form, where the overwhelming septic intoxication may cause death, as in scarlet fever, within twenty-four hours. Convulsions are common here. In the **abortive** form a typical onset is followed speedily by amelioration of symptoms and recovery. The temperature in some cases is distinctly intermittent, so that an **intermittent** type of cerebro-spinal fever is often spoken of.

Among sequelæ may be named optic atrophy, deafness, paralyses of spinal nerves, chronic hydrocephalus, chronic meningitis, aphasia, etc. Pneumonia is a not uncommon complication.

Differential Diagnosis. Tubercular meningitis has a more gradual onset, no rash, no herpes, rarely opisthotonos, usually tubercular history, or tuberculosis of some other organ.

Secondary Meningitis. Otitis media, mastoiditis, suppuration of ethmoidal sinuses, abscess of liver, gangrene of lung, or some other primary disease discoverable.

Typhoid Fever. More gradual onset, remittent temperature, tympanites, enlarged spleen, rose-spots, often diarrhœa; absence of herpes, petechial eruption, opisthotonos, paralyses.

DIPHTHERIA.

The diagnosis of faucial diphtheria rests mainly upon the local manifestations of the disease—*i. e.*, upon an examination of the throat. Redness and swelling, especially marked about the tonsils, are followed by the appearance of a whitish or grayish deposit, usually first upon one tonsil. This spreads until, in cases, not only both tonsils, but the soft palate, pharynx, naso-pharynx, nares, and larynx may be invaded. The membrane has an appreciable thickness, is somewhat firm, and so attached to subjacent tissues that attempts at its removal cause laceration and bleeding. Later it grows darker in color, often friable or soft, and emits an extremely offensive odor. The swelling of the throat may be extreme, the tonsils meeting in the centre. The adjacent cervical glands are usually enlarged. This local manifestation of the disease is the result of infection by the bacillus diphtheriæ or the Klebs-Loeffler bacillus.

Recently bacteriological examination of the membrane has been employed as a means of diagnosis in the doubtful cases, which are many, for at times the local evidence is slight or resembles so closely the simpler forms of throat inflammation —*e. g.*, follicular tonsillitis, scarlatinal angina—that without the detection of the specific organism a diagnosis is impossible.

Cover-slip preparations can be made by spreading a thin film taken with a sterile swab of cotton on a cover-glass, drying in the air, then over flame, and staining with gentian-violet or alkaline methyl-blue.

Should an examination with an oil-immersion lens show no bacilli, but only cocci, we may say that the specific organism of diphtheria is absent—we are dealing with some disease other than diphtheria. In case bacilli are present we must await the result of culture-experiments, unless experience has enabled us to recognize the diphtheritic organism and to distinguish it from others. The bacillus is about as long as the tubercle bacillus, non-motile, and characterized by the irregularity of its form. It may be perfectly straight, slightly bent, with rounded ends, or often seems to have within its irregularly stained structure a deeply stained granule, looking almost like a node upon the rod. Culture-experiments can be made with glycerin-agar in tubes and plates, or, better, with Loeffler's blood-serum and bouillon mixture. Upon the latter medium inside of twenty-four hours the colonies of diphtheria bacilli appear elevated, slightly grayish, with an opaque centre and slightly irregular border. They are of considerable size, and at first are moist. Inoculation of guinea-pigs can, if deemed advisable, be employed as a confirmatory test.

Dr. George H. Weaver, of the Bacteriological Laboratory of Rush College, kindly furnishes the following synopsis of the method of making a rapid bacteriological diagnosis of diphtheria:

Bacteriological Diagnosis of Diphtheria. **To obtain material for cultures:** Swabs are prepared by wrapping a small pledget of cotton about the end of a small, stiff steel wire, slightly roughened. The wire is cut at such a length that it will fit in a test-tube. The test-tubes containing the swabs are stoppered with cotton and sterilized for one hour at 150° C.

The patient is placed in a good light, the tongue depressed, and the cotton swab rubbed freely but gently against any visible exudate. If there is no exudate, or it is confined to the larynx, rub the swab freely against the mucous membrane of the pharynx and tonsils. Before the swab is replaced in the tube rub it over the surface of the solidified blood-serum, firmly, but not to break the surface.

Incubation of culture. The inoculated tube must be kept at from 35° to 37° C. for twelve to eighteen hours. This may be accomplished by the use of an incubator, or by carrying the tubes in an inside pocket or tying them in the axilla of the patient after protecting them with a suitable case.

Examination of culture. In from twelve to eighteen hours at 35° to 37° C. the bacilli diphtheriæ will have developed elevated white colonies; other bacteria present do not grow so much in so short a time. Cover-slip preparations are made from the colonies, stained two to five minutes with Loeffler's alkaline solution of methylene-blue, and examined with a one-twelfth inch oil immersion objective.

FIG. 8.

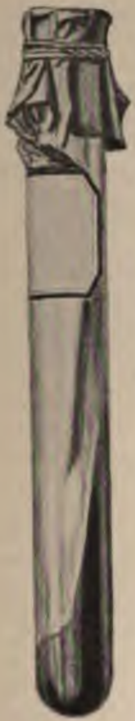
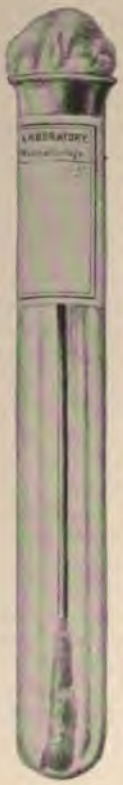


FIG. 9.



Test-tubes, with blood-serum and sterilized swabs, ready for use in diagnosis of diphtheria.

The bacilli diphtheriæ are recognized by their irregular or bizarre forms (clubbed at one or both ends, pear-shaped, etc.), and their irregular staining-reaction, staining deeply at one or both ends or at the middle, while the other parts are very slightly stained, or staining deeply and faintly in alternate transverse segments.

Determination of virulence. A pure culture of the bacillus diphtheriæ is obtained from the tube inoculated from the throat. A forty-eight hour bouillon culture of this at 37° C. is injected into a guinea-pig, in the proportion of ¼ to 1 per cent. of the body-weight.

Preparation of Loeffler's blood-serum mixture. One part of neutral beef-infusion bouillon containing 1 per cent. of grape-sugar is mixed with three parts of blood-serum. Place in sterile stoppered tubes. With the tubes in an oblique position so as to give a large surface when the media are solid, heat in the dry hot-air oven to a little below 100° C. until the serum is firm. On the three following days sterilize in the steam sterilizer for thirty minutes a little below 100° C.

Constitutional Symptoms. While the bacilli do not penetrate farther than the deeper layers of the pseudo-membrane, their action produces a chemical substance which, absorbed, results in certain pathological changes in the viscera and in constitutional symptoms, as varying in degree, however, as the local manifestations are variable in appearance and extent. Pain in the throat and in the muscles of the neck, while common, is at times trifling. Fever is even in some of the severer types of the disease low, though it may reach 104°. There are marked anorexia, weakness, pallor, frequently albuminuria. The pulse loses its fulness and becomes small and weak. The prostration of the patient is often strangely at variance with the trifling evidence of the disease in the throat. As the membrane sloughs there are improvement and convalescence, though paralysis of various nerves may make its appearance, now, or even days or weeks after apparent recovery. Nasal intonation, escape of liquids from the nose on attempts at swallowing, strabismus, are among the commoner evidences of post-diphtheritic paralysis. At times other nerves are involved, and inco-ordination of movements results. Sudden cardiac failure may accompany or follow diphtheria. Bronchitis, pneumonia, nephritis, endocarditis, are occasional complications or sequelæ of diphtheria.

Diphtheria of the nares causes obstruction to nasal respiration and an offensive, often bloody discharge from the anterior nares, irritating or excoriating the upper lip. The membrane may at times be seen creeping out from the nostrils. Nasal diphtheria is usually serious.

Laryngeal diphtheria can be diagnosed, aside from a laryngoscopic examination, which is often impossible in children, by the hoarse cough and voice, croupy respiration, and, as the obstruction becomes more and more marked, by evidences of imperfect filling of the lungs and impaired oxygenation of the blood—*i. e.*, by retraction of the soft parts on inspiration, by the cyanosis, restlessness, and feeble pulse.

The **differential diagnosis** of diphtheria is often a matter of extreme difficulty or of impossibility, save by careful bacteriological examination. Practically, we must treat all doubtful cases as diphtheritic. Follicular tonsillitis causes more headache, backache, higher fever, less prostration, and anæmia. The tonsils alone are involved; the whitish or yellowish spots correspond to the crypts of the tonsil, and can be readily and, if the manipulation be gentle, bloodlessly removed, showing the empty lacunæ. No firm, broad patch of membrane is seen.

In scarlatinal sore-throat there is usually a more diffuse, dusky red color, greater pain, a pultaceous rather than firm exudate, and distinct ulcers as the exudate disappears. The constitutional and skin symptoms, the peculiar tongue, usually render the diagnosis clear, though it must be remembered that genuine diphtheria and scarlatina may simultaneously affect the same person.

ERYSIPELAS.

Infection with the streptococcus erysipelatosus of Fehleisen produces a local dermatitis and lymphangitis, with marked constitutional symptoms. The infection-atrium may be a surgical wound, a traumatic break of the skin on any part of the body, but is oftenest, in the cases coming under the observation of the physician, a scratch or abrasion on the face, or in the nostril, often not discoverable.

Chilly sensations, or a pronounced chill, may usher in the disease. Malaise, headache, anorexia, rapid pulse are all found, as in other febrile conditions. Cerebral disturbances, especially delirium, are not uncommon. Locally, a spot of redness, gradually enlarging by a distinct border, and giving rise to a sensation of burning and itching, is seen. The inflamed area is swollen, tender, has a distinctly marked border, a glossy surface, and is of a dark, reddish hue. Reddish lines marking the inflamed lymphatics, which contain the parasite, can often be seen radiating from the erysipelatous area. The neighboring lymphatic glands may be enlarged. Vesicles and bullæ are at times seen, and infection of the deeper tissues may produce phlegmonous cellulitis. Pus is less frequently found, however, complicating facial erysipelas than erysipelas in other portions of the body. The disease spreads from the nose, usually over the face and scalp, disfiguring the face by the swelling produced, and may in certain cases invade the region of the chest and abdomen, constituting **erysipelas migrans.** The area first involved clears up in a few days, and the outer layers of skin are shed; often the hair falls out following the disease.

Relapses and recurrences are common. I have known of seven distinct attacks of facial erysipelas in three winters. With the abatement of the local evidences of inflammation there is improvement in the constitutional symptoms as well.

Among complications must be mentioned the extension of the disease to the pharynx and larynx, producing mechanical obstruction to swallowing and breathing by the accompanying œdema. Meningitis has proven a fatal complicating disorder. Albuminuria during the height of the attack is perhaps the rule. Genuine acute nephritis is not often met with. Death may occur from the severity of the septic intoxication or from intercurrent diseases. Pyæmia occasionally is seen. Usually facial erysipelas runs its course and terminates favorably inside of ten days.

Acute **eczema faciei** in children, with its rough skin, the involvement usually of both cheeks and of the chin, its afebrile course, can only be taken for erysipelas by the laity.

Chronic **erythematous eczema** of the aged itches, shows scratch-marks, is chronic, afebrile, the skin is not shiny; there is little swelling, and there are no vesicles.

Herpes zoster is unilateral, very painful; vesicles appear before there is much redness or swelling, and not later, as in erysipelas.

Simple dermatitis has little or no fever; some cause, as the application of some cutaneous irritant, can be found; the sharply defined and progressively advancing border of erysipelas is lacking; the purple color and smooth surface of erysipelas are rarely seen.

SEPTICÆMIA AND PYÆMIA.

These two classes of diseases ordinarily regarded as belonging to the domain of surgery come so often under the care of the physician, that it is important to say a word concerning their diagnosis, and especially as the physician is more likely to meet the masked cases than either the surgeon or accoucher.

Fermentation fever, absorptive fever, aseptic fever, or operation fever, as it is variously termed, is the fever seen after injury or operation. The greater the amount of tissue damaged, the greater the amount of blood and serum outside the vessel in the wound, the greater is the febrile paroxysm. Temperatures exceeding 104° are rare. Three days are seldom occupied by the febrile disturbance. Slight increase in pulse-rate, perhaps headache and consequent sleeplessness, are the only evidences of a departure from health.

Sapræmia. Dead tissue infected by putrefactive bacteria, and in such locality that absorption of ptomaines into the circulation can occur, gives rise to sapræmia; strictly speaking, an intoxication and not an infection. The parturient uterus, with the yet unexpelled secundines, the arm or leg the site of moist gangrene, or the gangrenous area in the lung afford good examples of this condition. A chill is occasionally the note of warning that there is trouble. The temperature varies according to the amount of toxic substances absorbed. Rapid and feeble pulse, early prostration and exhaustion, gastric and intestinal disorder, with perhaps delirium, dry tongue, and typhoid state are present in the severer cases. The odor from the necrotic mass usually gives the clew to the seat of the trouble. Unless there is prompt removal of the offending material death generally ensues.

Septicæmia. The entrance of organisms, usually the pus streptococci and staphylococci, into the circulation, and their multiplication there, with the production of toxins, give rise to a progressive train of symptoms known as septicæmia or blood-poisoning. The focus of infection may be a wound, as a post-mortem or dissection cut, or an apparently insignificant abrasion. The lesion may be hidden—*e. g.*, in the intestines. Before the days of Listerism this form of disease was common after surgical operations and after parturition, where the digitally diligent accoucheur was present on time. Lymphangitis and lymphadenitis can often be made out near the infection-atrium. Some of the severest cases may be ushered in with only chilliness, gradual rise in temperature, and increase in pulse-rate. In other cases a severe chill initiates the disease, and the temperature rises rapidly to 104°–105°. Nervous symptoms are usually marked, delirium, jactitation, restlessness, subsultus being often present. The fever is constant, somewhat irregular, but yet showing, as a general thing, morning remissions. The pulse gradually grows weaker and more rapid, and, preceding death, is often intermittent. Emaciation is marked, even in cases lasting only a week, and the face has a wan, pinched look seldom seen save in septic conditions. Sweats are common. Gastric irritability or diarrhœa may exist. Fatal cases rarely last longer than ten days. Milder cases, in which, by virtue of treatment or the germicidal power of the patient's blood, the bacteria are destroyed, or their products rendered inert, usually terminate by a gradual disappearance of the evidences of disease. It is rare that the sudden improvement is seen in septicæmia that is seen in sapræmia following the removal of the putrefactive focus.

Pyæmia. Pyæmia is rather a complication of a suppurative lesion, which may be located in any portion of the body, than an independent disease. The vessels in the vicinity of the suppurative focus become the site of thrombosis, and from this coagulum which is, it must be remembered, infected, fragments, large or small, as the case may be, become detached, and each embolus thus set free in the circulation becomes, at its point of lodgment in some distant vessel, as in the lung, kidney, liver, a new centre of suppuration.

The clinical diagnosis of pyæmia is made by a knowledge of:

1. **A suppurative focus.** This may be a visible surgical wound, or accidental trauma; a suppurative mastoiditis following middle-ear disease, the sequel of scarlatinal or diphtheritic angina; a pylephlebitis or hepatic abscess, perhaps secondary to intestinal ulceration; inflamed and suppurating hemorrhoids, or appendicular abscess; osteomyelitis, with or without the history of trauma; gonorrhœa, chancroids, with inguinal bubo; ulcerative endocarditis which, usually secondary, occasionally seems to be the primary suppurative trouble.

2. **Chills.** Malaise may precede the first chill, or a rigor may be the initial symptom. The chills are severe, often lasting an hour, and recur at irregular intervals for days or even weeks. At times their occurrence daily or on alternate days leads to the mistaken diagnosis of malaria. Sweats follow the chills.

3. **Fever.** Often during and immediately succeeding a

chill, the temperature is 103°–106°. Between the rigors there may be a complete intermission, though in the acute cases often only a remission of temperature. The pulse keeps pace with the temperature, though, as the disease progresses, it is found more rapid and weak.

4. **Formation of Metastatic Abscesses**, as evidenced by the local signs of disease and altered function of the part involved. Among the organs and tissues most commonly the seat of embolic infection may be mentioned the lung, spleen, kidney, liver, joints, pleura, peri- and endocardium.

5. **Emaciation**, loss of strength, often hæmatogenous jaundice, and toward the last mental dulness, with, perhaps, delirium. Subcutaneous and retinal hemorrhages or abscesses may be noted.

6. **Examination of the blood** in septicæmia and pyæmia will often show, by staining, micro-organisms, *e. g.*, the streptococcus. Septicæmia is very often mistaken for typhoid fever, and pyæmia is as often erroneously diagnosed as malaria. The irregularity of the chills, the primary and distant abscesses, and the absence of Laveran's organisms make a differential diagnosis not usually difficult. It is well to remember, too, what Osler has so well said, that "an intermittent fever which resists quinine is not malaria."

The forms of septicæmia in which no point of entrance for the bacteria is demonstrable during life are the most difficult to recognize. In some cases, even after death, no primary lesion is found. To such cases, which during life gave evidences of sepsis, often with a pyæmic element, as evidenced by chills and irregular temperature, the name **spontaneous** (Leube) or **cryptogenetic** (Jürgensen) **septico-pyæmia** has been applied. The more search is made before or after death the oftener some suppurating focus or some abrasion of the skin or mucous membrane is detected, through which the bacteria could have gained entrance to the circulation.

ASIATIC CHOLERA.

Infection with the comma bacillus of Koch produces in man an intense irritation of the mucous membrane of the intestinal tract, loss of epithelial cells, rapid osmosis of the fluid of the blood, and a most intense prostration and collapse. To this disease, epidemic in character, the name cholera is given, often spoken of as true, Asiatic or epidemic cholera, to distinguish it from cholera morbus, cholera infantum, etc.

There can be recognized in typical cases a stage of invasion, an algid stage, and a stage of reaction.

The **stage of invasion** is characterized by a sense of languor, epigastric pain, anorexia, and often, it is said, by a sense of impending danger and dread of the disease. Diarrhœa sets in, the stools, at first bile-stained and offensive, in the course of a few hours or days becoming more copious, inodorous, and of a rice-water character. The fluid that has escaped from the vessels contains epithelial cells enough to give it a slightly milky appearance, compared to rice-water. The stools are painless and are forcibly discharged. There is little or no fever. The pulse becomes weak and compressible, and the effect of the loss of fluid of the blood is shown by rapid emaciation, intense thirst, scanty or suppressed urine. Vomiting may be continuous. The muscles of the calves of the legs are painfully cramped.

The **algid stage** is but the continuation of the preceding. The temperature is subnormal, the skin cold, the breath cold. The pinched face, with sunken eyes, the wrinkled, cyanotic skin, sunken abdomen, hoarse whisper, rapid, feeble pulse, shallow respiration make a picture seen in no other disease. The patient's mind is often clear, though he may be restless and anxious, perhaps dull and apathetic. The vomiting may have ceased, and even the rice-water discharges become less frequent, apparently because of lack of material to be ejected. Nausea may be annoying. Death often takes place in this stage.

The **stage of reaction** shows the patient gradually becoming warmer, the pulse increasing in strength, the voice regaining its normal timbre, urine reappearing. Convalescence may be quite rapid and uninterrupted by complications; or the patient passes into a febrile state, marked by mental dulness, nervous prostration, gastric and enteric irritability—the condition known as cholera typhoid. From this he may gradually emerge or may succumb to exhaustion, or complicating affection such as pneumonia, diphtheria, nephritis, and uræmia. Nervous disorders, furuncles, eye affections, chronic gastric and intestinal catarrh may be sequelæ of cholera. In suspected cases the stools should at once be subjected to examination by a competent bacteriologist. It is unwise and unsafe for one not an expert to handle and pass bacteriological judgment upon the stools of these patients.

YELLOW FEVER.

This disease, endemic in the West Indies, occasionally during the summer months becomes prevalent as a severe epidemic along the coast in our Southern States. Newcomers to an infected district and those living unhygienically are most often attacked. It begins, as do so many of the acute infectious diseases, with a chill, rapid rise in temperature, headache, vomiting, and great depression.

The tongue is coated, the eyes reddened and watery, and complaint is often made of pain in the epigastrium, as well as in the back and muscles generally. Constipation is the rule. The urine, usually small in amount, is often albuminous. Convalescence may follow the continuance of these symptoms for a few days, or the amelioration of symptoms which then occurs (stage of remission) may be followed by a return of the fever and evidences of a renewed activity of the disease. There is now a yellow hue to the skin, which has given the disease its name. Vomiting is uncontrollable, and the vomitus is largely composed of dark blood, the dreaded "black vomit." An hemorrhagic tendency is often manifest at this stage. Scanty urine, albuminous and containing casts, may be the warning of impending uræmia. Death from exhaustion is common, and especially in those who have had the "black vomit." Few cases last longer than a week. Some, however, of a milder type go on to convalescence with no second paroxysm of the disease. Other cases, which might be termed abortive, last but a few hours or a day, recovery following rapidly, while in the most malignant cases a few hours are sufficient for the sudden onset with delirium or convulsions, vomiting, and death in collapse.

Severe and remittent types of **malarial fever** are positively recognized by the plasmodium in the blood. In these cases, too, there is rarely the hemorrhagic tendency, black vomit, or icterus. Herpes is common; delirium more often present. Urine rarely so early albuminous. Quinine modifies course of disease.

Acute yellow atrophy occurs oftenest in women in the pre- or post-partum state, and is characterized by the earlier occurrence of jaundice, the rapid change in the size of the liver, leucin and tyrosin in the urine. Fever is not so high here.

Relapsing fever. The spirilla in the blood, absence of jaundice and of black vomit will establish a diagnosis.

DYSENTERY.

There are recognized four clinical forms of dysentery, where the pathological findings are inflammation of the large intestine, ulcerative in character, and where the common symptoms are griping, abdominal pains, tenesmus, frequent, small, bloody mucous stools. These forms are the acute catarrhal, acute diphtheritic, amœbic, usually acute, and the chronic The etiological factor, probably microbic in all of these forms, is not yet clearly understood, and it is impossible to assign some of the cases clinically, at least, to their proper classes.

1. **Acute catarrhal dysentery** is at times ushered in with, and gives the symptoms of, an acute infectious disease. A chill or chilliness, preceded by premonitory malaise, may be noted, the temperature fluctuating between normal and 103°. The stools increase in frequency, lose their feculent character, and at the end of twenty-four hours become small in amount, nearly inodorous, bloody and stringy, or gelatinous from a large amount of mucus. The microscope reveals red blood-corpuscles, leucocytes, intestinal epithelium, and large epithelioid cells. Later, shreds of tissue may be seen even by the naked eye. The stools may be passed as often as every ten minutes, and there is an almost constant desire to bear down and strain—tenesmus—even though nothing passes the anus, or only a drachm or two. The griping pains caused by the peristalsis of the inflamed large intestine are so severe as, at times, to demand opiates for their relief. The patient may be prostrated, weak, and emaciated. The urine is scanty, the thirst extreme, the tongue at first coated, later red and dry. A natural tendency to recovery generally manifests itself by the ninth day, by the decreased frequency of the passages, the reappearance of fecal matter, and disappearance of blood, mucus, and shreds. The general condition of the patient likewise improves. Chronic dysentery or fatal exhaustion may unfortunately result. The diagnosis is usually readily made by the small, frequent stools, the blood, mucus, and shreds they contain, the colicky pains, and the tenesmus.

2. In some cases of dysentery there are found, post mortem, large areas of necrosis in the colon; this is known as **diphtheritic** or gangrenous dysentery.

Clinically this form causes earlier, greater prostration, at times delirium, weaker pulse—in short, a typhoid condition. Grayish and black pieces, large and small, of the necrotic membrane may be found in the stools, which are less frequent, painful, and bloody than in the simple catarrhal form, but having an offensive, gangrenous odor. The affection may occur as a secondary process in the course of faucial diphtheria, pneumonia, or simple catarrhal dysentery, or it may be primarily intestinal. Fatal results are not uncommon.

Amœbic dysentery is the form prevailing in the tropics often as an epidemic. Numerous sporadic cases have been reported in temperate climes. The amœba coli, probably rightly regarded as the cause of the infiltration of the submucosa with consequent ulceration and undermining of the mucosa of the large intestine, is so uniformly found in the stools of these cases as to be a certain means of diagnosis. The stools must be submitted to careful microscopic examination in all cases of subacute or chronic dysentery or suspiciously intractable diarrhœa for the amœba, and especially in the case of patients who have been sojourning in tropical climates. Symptomatically, we may find about the same clinical picture as in simple catarrhal dysentery, though the stools may not be so frequent, so bloody, or attended by so much pain and straining.

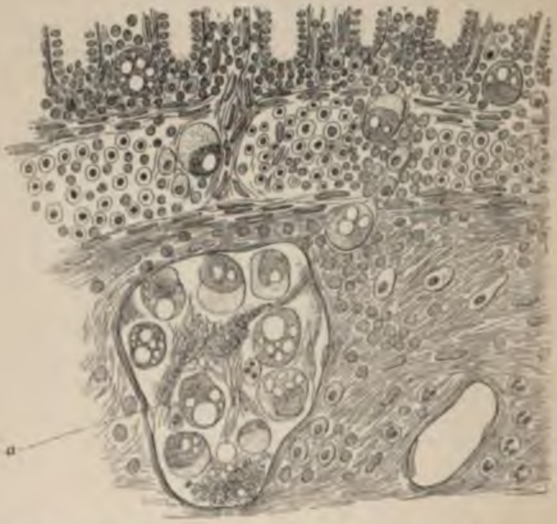
Slight fever, asthenia, and emaciation accompany the progress of the disease, which may last twelve weeks, end in recovery or death, or in apparent recovery and subsequent recurrence. The stools of amœbic dysentery while early much like those of the catarrhal form, later are more fluid and yellowish-gray in color. Amœbæ are found in the stools, especially when the stools are most frequent. The organism is about twice or thrice the size of a red blood-corpuscle, unicellular, nucleated, has an outer clear zone, the ectosarc, and a granular inner zone, the endosarc. One or more clear vacuoles are usually seen. Amœboid movements can be observed for some time after the organism is placed on the slide, if the temperature of the room be not too low. For preservation they can be stained with eosin and hæmatoxylon. (Fig. 10.)

In connection with tropical abscess it must be remembered that abscess of the liver, at times causing right sided empyæma or pulmonary abscess, is frequently observed. Amœbæ have been found in these abscesses with great uniformity.

For further details as to amœbic dysentery and liver abscess the student is referred especially to the article by Councilman and Lafleur, *Johns Hopkins Hospital Report*, vol ii., and to Kartulis, *Virchow's Archiv*, Bd. cxv. S. 97.

Constant diarrhœa, perhaps alternating with constipation, the tendency to exacerbations when blood, mucus, and pus are passed, exhaustion, and often very marked anæmia and emaciation, especially with a history of preceding acute dysentery, enable us to make a diagnosis of **chronic dysentery**, though the dividing line between this and some cases of chronic diarrhœa cannot be sharply drawn.

FIG. 10.



Section of intestinal wall near amœbic ulcer, showing amœbæ in the mucosa, submucosa, and in the lymphatics just beneath the muscularis mucosa. (COUNCILMAN.)

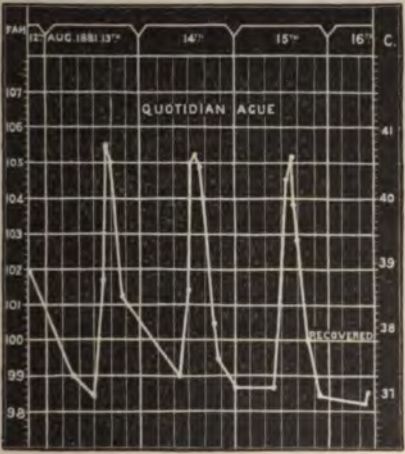
MALARIA.

Malarial infection presents to us for diagnosis four clinical forms: **intermittent, remittent, pernicious,** and **chronic.** In general, it may be said the evidence of the disease is furnished presumptively by (*a*) known *exposure* to infection by residence in a malarial locality; (*b*) the *periodicity* of the malarial paroxysm; (*c*) the yielding of the disease to *quinine*. Conclusive evidence is the discovery of the **malarial parasite** in the blood.

In this country the regions known to be malarial are less numerous than formerly. Still in the Southern and Southwestern States, and especially along river bottoms and in swampy regions, are places where newcomers particularly are liable to be infected with the malarial organism. There is more danger at night than in the day, and more liability to infection in the case of those sleeping near the ground than in those sleeping higher up. More cases are met with in the spring and autumn than in summer and winter. Warmth and moisture seem to favor the activity of the organism.

Before the discovery of the parasite the periodicity characteristic of acute malarial diseases was the mainstay in diagnosis. A typical paroxysm, as in intermittent, consists of a chill, usually severe, and attended by a rise in temperature, the fever continuing for a few hours, and defervescence, usually sudden and attended by profuse sweating. Premonitory malaise and accompanying headache, vomiting, pains in the various parts of the body are often present.

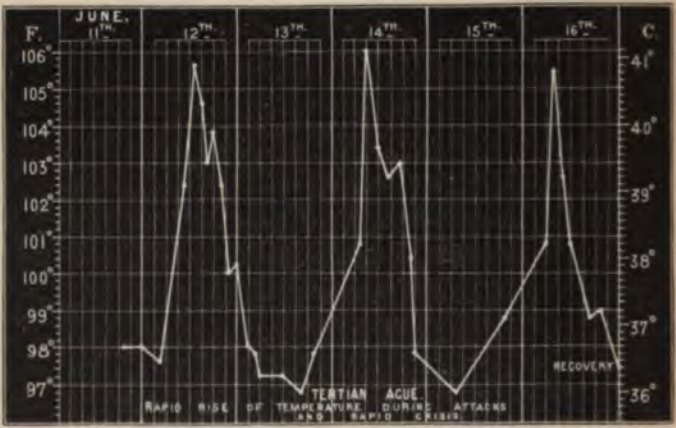
FIG. 11.



Quotidian malarial fever. (FINLAYSON.)

With the sweating there is amelioration of all unpleasant symptoms, and during the intermission—*i. e.*, the period of apyrexia—no complaint is made by the patient, who may be busy about his everyday work. The *interval* or time intervening between the beginnings of two consecutive paroxysms varies, but is usually regular, and it is this *regularity* of the paroxysms that distinguishes the chill, fever, and sweating of malaria from that of such diseases as pyæmia, ulcerative endocarditis, etc. Where the interval is twenty-four hours the fever is spoken of as quotidian; where forty-eight hours, as tertian; seventy-two hours, as quartan.

FIG. 12.



Tertian malarial fever. (FINLAYSON.)

In the **remittent** type of malarial fever, instead of a true intermission or apyrexia, there is a marked remission—comparative apyrexia; the paroxysms, while usually distinctly marked, lack the severe chill and the sudden complete defervescence of the true intermittent.

The disappearance of all manifestations of the disease under the administration of quinine is presumptive evidence of the plasmodial character of the disease.

Some forms, however, are very resistant against quinine, and in some of the remittent, pernicious, and chronic types of the disease the plasmodium is to be sought for not alone as a pathological curiosity, but as the only certain proof of the existence of the disease. The positive diagnosis—at least, early diagnosis—of some forms of malaria is impossible without the detection of the plasmodium.

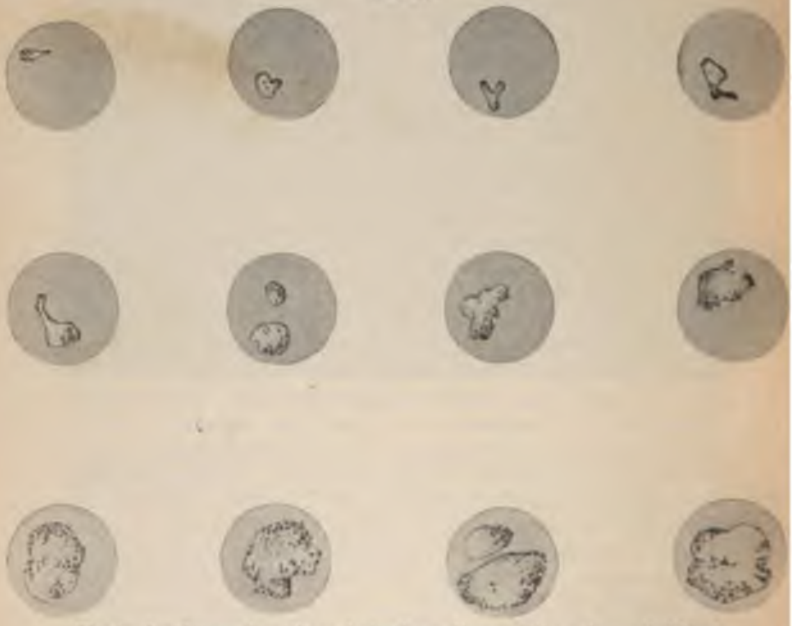
There are two general methods of examining malarial blood: the examination of (*a*) the fresh specimen; (*b*) the stained specimen. The following methods are among the simple ones of use in diagnosis. Various modifications are described, especially where the various phases in the life-history of the plasmodium are to be observed.

For clinical use, where a rapid diagnosis is desirable, a drop of blood can be placed under the microscope and examined immediately. Minor details are here all important. The finger or lobe of the ear from which the blood is drawn, the needle or fine lance-shaped scalpel with which the skin is punctured, the thin cover-glass (No. 1), and the slide must all be scrupulously cleansed with water, ether, and absolute alcohol. It is better not to squeeze the part from which blood is obtained, but to make a free enough incision so that blood flows without squeezing. The cover should be held by a clean forceps, and not touched by the finger. A small drop of blood on the slide quickly covered by the glass should be placed under an oil-immersion lens.

We may recognize for clinical purposes an endoglobular form of parasite, an ectoglobular, and the crescent forms. Each red corpuscle is to be carefully examined for mobile, clear, hyaline, or faintly whitish bodies of various shapes and sizes, at times quite filling the red globule, and which often contain pigment, black or brownish-red. This pigment, often clustered in several groups in the organism, is seen to be in active motion, aptly compared by Laveran to the dancing of grains of sand in boiling water. It resembles the Brownian movement, but is believed to be due to the movements of the protoplasm of the plasmodium. The plasmodium may, under the eye of the observer, change its shape. Keeping the slide at an equable high temperature, 80° to 100°, favors these amœboid changes. Just preceding the paroxysm of intermittent the plasmodium is large, often nearly or quite filling the degenerate red corpuscle, and the pigment—derived from the corpuscular hæmoglobin—is distributed among several lobes or segments of the organism. This rosette-form marks the culmination of the life-history of the plasmodium. It soon undergoes segmentation; the separate small segments are set free in the blood, enter other red corpuscles, and gradually during the interval grow at the expense of their host, the red globule, until the organism is again "ripe," when the same process is repeated.

Ectoglobular forms, all perhaps derived from the endoglobular, are seen in all forms of malaria, usually smaller, frequently with one or more filaments that by their active motion may disturb the red blood-disks, and in that way call the attention of the observer to their presence. Loose filaments are occasionally met with. Pigment-containing bodies curved upon themselves, the *crescents* of Laveran, at times within the red corpuscles, where perhaps they always originate and the size of which they frequently surpass, at times outside the corpuscle, are seen in some intermittents. In remittent fever, in the pernicious form, and in malarial cachexia, the plasmodium does not reach the size of that of the typical intermittent; the "small plasmodium" of the Italians is found. Crescents are also more abundant.

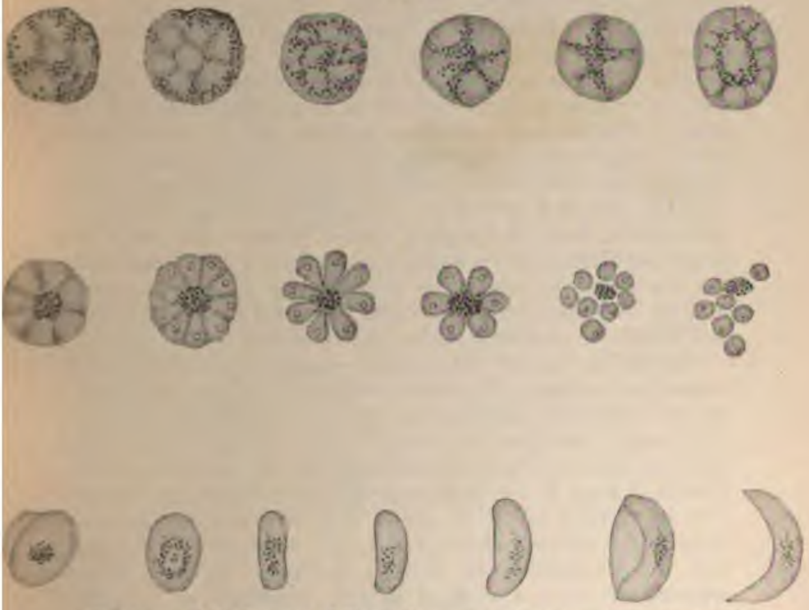
FIG. 13.



The twelve figures show the malaria plasmodium. It is a pale amœboid body inside the red corpuscle. It increases in size at the expense of the corpuscle. In the last four of the twelve it is enlarged, and contains pigment-granules derived from the hæmoglobin.

To stain malarial blood, obtain a drop carefully as before directed upon a cover-glass, which is held by a forceps. With a second cover-glass, likewise held by forceps, the drop is quickly and gently thinly spread over the opposing surfaces of the two glasses, the covers separated, and the specimens allowed to dry in the air. They are then fixed by immersion for fifteen minutes to one hour in a mixture of equal parts of absolute alcohol and ether, and then stained for three minutes in a ½ per cent. solution of eosin in 70 per cent. of alcohol, which, before using, had better be diluted one-half with water. The cover is then air-dried or dried between filter paper, stained for thirty to sixty seconds in a saturated solution of methylene-blue (diluted one-half with water), and examined mounted in balsam. Various combinations of eosin and methyl-blue are advised; but this method is perhaps as simple

FIG. 14.



The figures of the first row of Fig. 14 show progressive stages in the process of cleavage of the plasmodium, and shifting of the pigment-granules. In the second row the process of cleavage is seen to be completed, and final isolation of the spores has taken place. The dark granules are pigment-granules. The last row shows oval parasites, Laveran's corpuscles observed in atypical cases of malaria. —Golgi, "Studien über Malaria," *Fortschritte der Medicin*, Bd. iv., Tafel, iii. (From "Musser's Diagnosis.")

as any, and admits of as easy application. By this staining, the red corpuscles are stained red and the nuclei and plasmodia blue. The oxyphilic granules of the leucocytes show their affinity for eosin by taking its brilliant red stain. For rapid work an immersion for ten minutes in the fixing-solution will permit an examination of the stained specimen inside fifteen minutes, making this method practically applicable as a bedside means of diagnosis. Plate I.

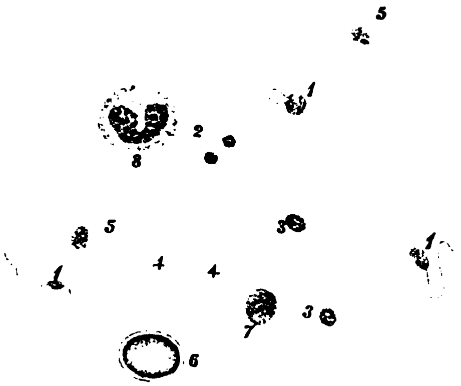
Hochsinger (*Wiener med. Presse*, 1891, No. 17) advises the following method, which will be found very reliable and simple: Fix the air-dried cover-glass preparation by immersion for twenty to thirty minutes in the absolute alcohol and ether. The staining solution is made by taking three ounces of saturated aqueous solution of methyl-blue, adding a few drops of absolute alcohol, and then seven and one-half grains of eosin dissolved in water. Sterilize by boiling for thirty minutes and keep in a tightly corked bottle. For use a few drops are filtered into a watch-glass and the solution warmed; the cover-slip preparation is floated on it for ten to twenty minutes and then washed in water, dried between filter-papers and then over an alcohol-lamp, and mounted on balsam.

Jaksch (*Klinische Diagnostik*) recommends the following method of staining the fresh, unfixed specimen of blood: To a 0.6 per cent. salt-solution (normal salt-solution) enough of a watery solution of methyl-blue is added to make the mixture moderately blue. This is filtered, the filtrate sterilized, and kept in sterile bottles, preferably several small ones. The finger from which the blood is to be taken is cleansed, a drop of the staining-fluid placed over the end of the finger, and the puncture made through the drop. A drop of the mixture of blood and fluid is then examined as described above. The plasmodium stains blue, and is more readily detected outside the red corpuscle than when unstained, and its boundaries in the red globule are easier made out by the different shade of blue imparted to the plasmodium. If the cover be surrounded by paraffine or vaseline, the rapid drying of the specimen is prevented.

Intermittent fever, ague, or chills and fever, is, in temperate climates, the commonest form of malaria. Typical cases are characterized by the occurrence, at regular intervals, usually of twenty-four or forty-eight hours, of paroxysms as before described—chill, fever, and sweating. Headache is common at such times. There is often vomiting; constipation is the rule. In the interval the patient may seem well, though

PLATE I.

Fig. I.



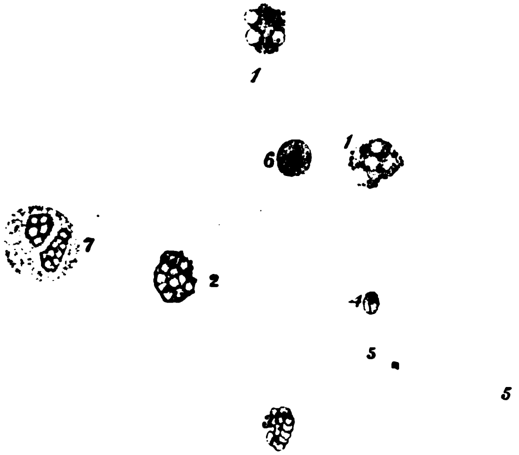
Malarial blood stained five hours after the fever has disappeared. (Plehn.)

1. Ectoglobular young parasites.
2. Endoglobular young parasites.
3. Dead parasites.
4. Indentations in the blood corpuscles (Quincke), which have been mistaken for parasites.
5. Diffuse methyl blue staining of the central part of the corpuscle, likewise easily confused with the parasite.

6 and 7. Leucocytes (large and small lymphocytes).

8. Eosinophile.

Fig. 2.



Malarial blood taken just before the chill. (Plehn.)

1. Differentiation of the protoplasm in the body of the parasite indicating the beginning of sporulation.

2 and 3. *Sporulation forms.*

4. Two free spores held together by a remnant of protoplasm.
5. Same as Figs. 1, 4.
6. Leucocyte (Lymphocyte).
7. Eosinophile.

frequent recurrences of the paroxysms lead to continued malaise, anorexia, anæmia, with its train of symptoms. The chill of intermittent is ordinarily a severe one: the skin seems cold, the face is pale, the lips blue, the teeth chatter. The temperature begins to rise, often even before the time of the chill, and remains high for from one to several hours. During the fever the skin is hot, the pulse bounding, the patient prostrate. Atypical paroxysms, known as dumb ague, are met with, where there is only a chilliness, slight rise in temperature, but where the headache, vomiting, pains, periodicity of the abortive attack give evidence of its malarial character, confirmed by the quinine test and by the discovery of plasmodia.

Remittent fever is commoner in warm than in temperate climates. A chill often ushers in the disease. Anorexia, severe throbbing headache, and gastric disturbance are common features; the tongue is heavily coated, the bowels are usually constipated, the spleen enlarged. The urine is scanty, acid, may contain a trace of sugar. It is rarely albuminous. The temperature is distinctly remittent, high in the evening, with a morning remission of from two to five degrees. All symptoms are usually aggravated in the afternoon—*i. e.*, we have an attempt at a malarial paroxysm. If uninfluenced by quinine, we often find by the fourth day a fever of the continued type, the remissions being slight. A tendency toward spontaneous recovery, which often occurs about the tenth day, shows itself by a marked morning drop in temperature. Labial herpes is almost as common in this fever as in pneumonia.

Pernicious forms of malaria are seldom seen except in tropical countries. Here, apparently from an inability of the excretory organs to eliminate the chemical poisons, the result of plasmodial infection, there is a severe systemic intoxication, such that patients sometimes die in the first paroxysm. Usually, however, a fatal result is rarely reached before a second or third attack. Intermittent and remittent forms of the pernicious type are described. The chills are usually marked, the temperature high, and there are evidences of congestion of various internal organs, with great prostration, at times approaching collapse or even the algid stage of cholera. In these cases temperature may be even subnormal. In some cases coma or delirium may be prominent. Again, vomiting and purging, intense thirst and restlessness, rapid, feeble pulse, impaired respiration, may be the symptoms. This latter form is often spoken of as the gastro-enteric form of the disease. A hemorrhagic tendency is noticed in many cases, and in some is so prominent as to cause the disease to be spoken of as **hemorrhagic malarial fever.** In addition to the chill, prostration, etc., previously mentioned, there is the passage of bloody urine, at times bloody fecal discharges. Purpuric lesions are common and hæmatogenous jaundice may develop.

Malarial cachexia is a term applied to a chronic malarial infection. Repeated and especially untreated attacks of malarial fever may lead to a condition in which there are more or less weakness, disturbed digestive function, a muddy-white color of the skin and enlargement of the spleen and liver. At times neuralgic pains are complained of. Slight rises in temperature are perhaps noted. Blood examination shows anæmia often marked, frequently loose pigment in the blood, and the small plasmodium and the crescents. The diagnosis must rest mainly on the history of preceding malaria, the enlargement of the spleen, and the anæmia with all its symptoms —dyspnœa, dizziness, including at times hemorrhage.

The discovery of the plasmodium by Laveran has made the differential diagnosis no longer a matter of uncertainty, yet it is well to call to mind the fact that malaria is often taken for some other disease, and *vice versa.*

Chills, fever, and sweating, it must be remembered, may occur in many infectious diseases. Recurrent chills may for a time, by their periodicity, simulate ague. Thus in pyæmia, ulcerative endocarditis, some cases of gall-stones, concealed suppurative processes, florid phthisis, etc., the clinical manifestations may be very deceptive. Especially when quinine fails to cure should we doubt the malarial character of the disease.

Remittent and typhoid are easily confused. The examination of the blood is the only sure means of diagnosis. Yet we may remember that in typhoid there are usually a more gradual onset, less clearly defined afternoon exacerbations, more tendency to tympany and diarrhœa, and usually by the end of the first week the rose-colored eruption. Herpes is extremely rare in typhoid, and there is, I believe, less tendency to sweating until late in the disease.

TETANUS.

This malady seldom presents difficulties in diagnosis, as there is a history of a wound or injury which at once puts us on guard as to the possibility of the existence of wound-infection by the bacillus of tetanus. It is to be remembered, however, that the wound may be slight and easily overlooked unless carefully sought for; it may be in the cervix uteri, following parturition, or in the umbilicus of the newborn infant.

Symptoms commonly make their appearance in from seven to fourteen days after infection. A stiffness is noted in the movements of the jaw, the masseter muscles are found in a state of tonic spasm. Soon other muscles of the face and neck are involved, the face has a rigid, fixed look, presenting grinning features; the neck is stiff. Other muscles become involved in tonic spasm, the progress being from above downward. Opisthotonos may be present. General clonic convulsions may occur, causing intense pain to the conscious sufferer. As in strychnia-poisoning, a very slight stimulus suffices to produce convulsions. The spasmodic contraction of the diaphragm is shown by the dyspnœa and the pains in the lower part of the chest. The skin is moist, the bowels constipated, the urine suppressed or retained. Continued wakefulness, anxiety, pain, recurring convulsions may lead to death from exhaustion. Death often occurs during a convulsion. The prognosis is grave in the extreme.

Lockjaw or trismus occurring from causes other than tetanus is generally readily recognized. It is not to be forgotten that in mumps, in rheumatism, or synovitis of the temporo-maxillary articulation, in various forms of sore-throat, especially in tonsillar abscess, there is often more or less stiffness of the muscles of the jaw. Hydrophobia, tetany, and the stiffness of the neck associated with cerebro-spinal meningitis must not be confounded with tetanic trismus, nor must epilepsy or hysterical manifestations. In the case of tetanus the history of trauma, the short incubation, the trismus followed by rapid and progressive involvement of other muscles lower down, the risus sardonicus, the convulsions, with the absence of the confirmatory signs of other diseases, as the swelling of the parotid in mumps, involvement of other joints in rheumatism, swelling, redness, and pain in the throat in sore-throat, headache, delirium, fever, herpes, etc., in cerebro-spinal meningitis, make the diagnosis easy.

The contrasting and differentiating points between **tetanus** and **strychnia-poisoning** may be briefly summarized as follows:

TETANUS.	STRYCHNIA-POISONING.
1. History of trauma; usually the presence of a visible wound.	1. History of ingestion of drug.
2. Symptoms after several days.	2. Symptoms after a few minutes or an hour.

3. Involves first muscles of the jaw and neck, then progressively the muscles of the lower part of the body; the arms and hands affected late.	3. Muscles of the extremities involved early, or general convulsion involving all the body muscles.
4. Spasms, tonic from the first; convulsive seizures late and gradually severer.	4. Spasms, clonic from the first, and severe.
5. Persistent muscular rigidity even in the interval.	5. Muscular relaxation in the interval.
6. May last for days.	6. Never more than a few hours.
7. Bacillus may be found in the wound.	7. No organism.

RABIES—HYDROPHOBIA.

The incubative period varies between forty and sixty days, being shorter in children, in the case of extensive or deep laceration or wound of the skin, and where the bite of the rabid animal is on an exposed part of the body, as the face or hand, the teeth before entering the flesh not passing through the clothing. More rarely there is noted a much shorter or longer period of incubation. The wolf and cat, as well as the dog, can communicate the disease. There are three stages in the clinical history.

Premonitory stage. Usually there is an itching or burning sensation in the scar, which may appear of a brighter color than before. The patient is nervous, despondent, dreads some impending trouble. There are anorexia, slight rise in temperature. Slight pharyngeal constriction may be present.

Furious stage. Spasmodic contractures of the laryngeal and pharyngeal muscles are excited by very slight causes, a sudden noise, a draught of air, an attempt to swallow, or even the suggestion of swallowing. At the times of these paroxysms the patient may have slight or marked convulsive movements, may become maniacal, dyspnœa may be extreme, there is often salivation. The respirations in the intervals are often hurried, the mind clear, the temperature usually slightly elevated.

Stage of paralysis. After twenty-four to seventy-two hours muscular relaxation and coma ensue, death occurring in about twelve to twenty-four hours, usually by gradual or sudden heart-failure.

The diagnosis must depend largely upon the knowledge of a previous bite by a dog, wolf, or cat, especially if the animal has shown rabic symptoms, the long incubation, and the premonitory nervousness, dread, local scar symptoms, and later the pharyngeal spasms.

Spurious or hysterical rabies can usually be readily recognized by careful examination of the patient and history of the case.

ANTHRAX.

This disease occurs especially in workers among animals or their products, *e. g.*, stablemen, wool-sorters, workers in horse-hair, tanners. The bite of an insect may inoculate man or the lower animals.

At the point of infection, usually the face or hands, an itching spot soon shows a reddish papule that increases in size, becomes vesicular and pustular, the contents being discharged, and a dark-brown or coal-black (**anthrax**, a coal) centre marking the spot of greatest inflammatory activity. A wide area of brawny, infiltrated tissue surrounds this eschar, involving not only the face (where infection most frequently occurs), but even the neck and the arm. The neighboring lymph-glands are swollen. Examination of the fluid contents of the vesicle, or tissue from the inflamed area, will show the spore-containing anthrax bacillus, several times longer than a red blood-corpuscle, easily staining by most of the aniline reagents.

Within a few hours to a few days general infection occurs, and there are symptoms of sepsis, at times mild, but too often severe, and followed by fatal results. The splenic enlargement has given the disease the name of splenic fever. Bacilli can, late, be found in the blood.

Where there is infection through the gastro-intestinal tract the symptoms may develop suddenly and resemble those of poisoning,—vomiting, purging, muscular pains, dyspnœa toward the end, convulsions, and collapse. Here the diagnosis, as in the form where infection is by way of the respiratory tract, must rest upon the knowledge of possible infection, exclusion of other diseases, and the detection of the bacillus anthracis in the blood. Hemorrhages from the mucous membranes or into the subcutaneous tissue may occur. Cutaneous abscesses are also observed at times.

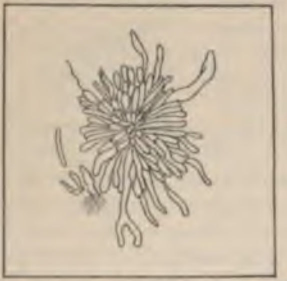
The diagnosis depends upon the knowledge of the patient's occupation, the rapidly developing pustule, the central brown or black eschar, the infiltrated areola, general symptoms, exclusion of other diseases, and the microscopic, culture, and inoculation proof of the presence of the bacillus anthracis.

ACTINOMYCOSIS.

Infection with actinomyces is oftenest through the mouth, as through a carious tooth. The lung, intestine, external ear, may be primarily affected, and metastatically other organs.

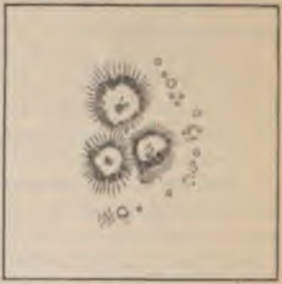
Chronic febrile, and perhaps pyæmic symptoms, at times amyloid degenerations, mark the course of the disease. In the pus from the discharging sinus under the jaw, or in the purulent expectorate in bronchial or pulmonary actinomycosis, fine yellowish particles can be seen with the naked eye. Under the microscope the characteristic ray fungus is seen. This is the only sure means of diagnosis. Where there has been exposure to the disease, as it occurs in the lower animals (lumpy jaw of cattle), the suspicion is at once aroused of the possibility of actinomycosis.

FIG. 15.



Actinomycosis. Sulphur-colored granule × 1100. (ISRAËL.)

FIG. 16.



Actinomycosis. Granule compressed × 300. (ISRAËL.)

GLANDERS.

Glanders is occasionally inoculated into man from horses through an open cutaneous or mucous wound.

In the acute form there are headache, fever, general pains. The wound has the angry look of an infected wound, and is sometimes surrounded by a diffuse dermatitis. A papular eruption appears within a week, the papules gradually being transformed into vesicles or pustules that upon rupture give rise to a purulent discharge. A fetid muco-purulent discharge occurs from the nose, which becomes reddened, painful, swollen. Death follows in from one to four weeks from the beginning of the disease.

In chronic glanders the ozæna may be absent. Ulcers form on the hand, face, and other parts of the body. Suppurative arthritis may be present. Cough, bloody expectoration, emaciation, fever may make the disease appear somewhat like pulmonary tuberculosis.

DISEASES OF DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Acute catarrhal stomatitis, found oftenest in children or in those weakened and debilitated by other disease, is produced by mechanical, chemical, or thermic agents, or by the extension of inflammation from the larynx or pharynx. Not infrequently quite severe types of acute stomatitis are met with in patients sick with an acute infectious disease, as typhoid or measles. The period of the first dentition is a favorite time for the appearance of stomatitis.

Dryness, redness, and swelling of the mucous membranes of the mouth, with a flabby, coated tongue, are followed by an increase in the flow of saliva. Pain may be so severe as to render mastication difficult. The mouth tastes bad, there is an offensive odor to the breath, and often in children slight fever and constitutional disturbance are present.

ULCERATIVE STOMATITIS.

In this form the inflammation is first seen at the edge ot the gums of the lower jaw. The gum is swollen, reddened, painful, easily bleeding. Soon an ulcer with a grayish-yellow base and rapidly extending, makes its appearance, the neighboring glands become enlarged and tender, the tongue grows foul and heavily coated, salivation is marked, and the odor of the breath inexpressibly offensive. The teeth may become loosened in their sockets, and the ulcerative process may even be so extensive as to cause necrosis of the jawbone. The patient, from failure to eat and sleep, from impaired digestion, may grow weak and exhausted. Mild septic symptoms in the shape of febrile disturbance are occasionally met with.

Mercury produces a stomatitis ushered in by a metallic taste in the mouth, pain on bringing the teeth together, a feeling as though the teeth were too long, swelling of the gums, salivation, and, in bad cases, phenomena not distinguishable from those of ulcerative stomatitis.

Ulcerative stomatitis is rarely met with among the healthy and well-nourished. But among the feeble with unsanitary surroundings, as in jails, camps, public institutions, it is not rarely seen, sometimes in the form of a mild epidemic.

APHTHOUS STOMATITIS.

In children, and even in adults, especially those suffering from general diseases or gastro-intestinal disorders, a form of stomatitis is met with known as aphthous stomatitis, often by the laity spoken of as "canker sore-mouth." Probably from some yet unrecognized microbic cause there is a localized inflammation in the superficial and middle layers of the mucous membrane of the mouth, with a fibrinous, grayish or whitish exudate. Vesiculation is said to be the earliest phenomenon, but it is certainly very rare clinically to demonstrate a vesicle. A whitish spot, from the size of a pin-head to that of a dime, with a red areola, is seen on the edge or under the surface of the tongue, between the tongue and the cheek, or just inside the lips. This aphthous patch is usually very slightly depressed below the surface of the mucous membrane. It causes great pain on talking or mastication. Salivation may be profuse. As many as a dozen or even more of these little patches may be seen, sometimes even on the tonsils and back in the pharynx.

This condition is readily distinguished, as it must be, from bits of coagulated milk clinging to the cheeks, from ulcerative stomatitis, from thrush, and from the mucous patches of syphilis.

THRUSH.

The development of the yeast fungus, **saccharomyces albicans** or **oïdium albicans**, upon the mucous membrane of the mouth gives rise to the condition known as *thrush*, söor, or muguet, so often seen in nursing-children where there is improper care of the mouth, the nursing-bottle, or the nipple, and in adults afflicted with some depressing disease, as tuberculosis. The oral mucous membrane is dotted here and there with a whitish deposit of an appreciable thickness that is removed with difficulty, and which, left to itself, usually grows until several of the original patches have become confluent. The deposit when removed shows a roughened base.

The microscope shows the parasitic nature of the disease, a bit of the whitish deposit revealing the interlacing and branching filaments, with budding cells developing at their ends.

NOMA.

Noma is really a surgical affection, rarely developing in children with sanitary surroundings. It is occasionally seen following mealses. It usually begins on the inside of one cheek as a swollen, inflamed area, soon developing into a spreading ulcer with brawny induration about it. There is rapid extension of the necrosis, mucous membrane, muscle, skin, bone giving way before it until death from hemorrhage, exhaustion, or sepsis occurs.

Acute catarrhal.	Ulcerative.	Aphthous.	Parasitic.
1. Often in healthy from mechanical, chemical, or thermal cause.	In poorly nourished or due to mercury, lead.	In teething-children, cachectic patients, or where there is gastro-intestinal disorder.	Usually in neglected nursing-infants, or in persons debilitated from disease.
2. No localization; entire mouth inflamed.	Begins on lower gums, which are soon ulcerated.	May be but one affected area or several; little swelling.	Usually many small spots increasing in number and size.
3. General redness and swelling; no ulcer.	Red, swollen, bleeding gums, ulcers with dirty, grayish-black base; teeth loosened, even bone-necrosis.	Depressed whitish area with narrow red areola.	Whitish elevated area; no areola. Parasite discoverable.
4. No constitutional disturbance unless previously existing.	May be weakness and exhaustion.	Rarely constitutional disturbances, unless previously existing; often constipation.	May spread to throat and œsophagus, and become serious.

The various forms of stomatitis are in most cases readily recognizable. They all manifest the common symptoms of the sense of heat and pain in the mouth, rendering nursing or mastication difficult, and causing restlessness and irritability in children, and even in adults. The flow of saliva is usually increased. In catarrhal stomatitis there is no local lesion, but a uniformly inflamed mucous membrane. Ulcerative stomatitis begins on the lower gums, is attended by bleeding, extremely fetid breath, often loosening of the teeth. Aphthous stomatitis has a whitish appearance, the affected area is often distinctly depressed below the surface of the mucous membrane, and there is a red areola. The parasitic form is elevated, has no areola, shows the parasite. In noma the brawny tissue, rapid gangrene, and evident destructive character of the disease call attention to its nature. Syphilitic mucous patches, diphtheria, bits of coagulated milk are also to be differentiated from the above-described forms of stomatitis.

DISEASES OF THE SALIVARY GLANDS.

SALIVATION.

Salivation is in reality but a symptom. It is met with as a reflex phenomenon in affections of the cranial nerves, in disease of the medulla or pons, and in some of the infective diseases, especially smallpox. It may be excessive during pregnancy or sexual excitement. Mercury, iodine, jaborandi, and other drugs may cause ptyalism. The frequent necessity of swallowing or expectorating, to avoid the dribbling of the saliva over the chin, is the annoying accompaniment of this troublesome affection.

XEROSTOMIA.

Xerostomia, or diminution of the salivary secretion, is found in fevers, and sometimes, through some not clearly understood nervous mechanism, the condition assumes the proportion of a disease, causing the mouth to become dry, parched, the mucous membrane red, and mastication and articulation difficult.

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Usually in acute catarrhal inflammation of the pharynx there is found also an involvement of the tonsils and often of the larynx and nares. Exposure to cold, the rheumatic diathesis, acute infectious diseases, are the common causes. The onset may be marked by a chill or by chilly sensations. Pain on swallowing, slight enlargement of the adjacent glands, in the rheumatic form especially stiffness of the muscles of the neck, call attention to the throat, which is found uniformly reddened, with engorged vessels and more or less swelling. The initial dryness is succeeded by increased secretion. Hoarseness, nasal discharge, deafness may accompany the disease if the larynx, nares, or Eustachian tube are at the same time affected. Fever may run to 103° or over, but usually subsides within forty-eight hours. The pulse is full and increased in rapidity. Often there are much muscular soreness, pain in the back, "aches in the bones," and a general discomfort and prostration out of proportion to the seemingly trivial affection.

Where the tonsils are chiefly or solely involved the affection is known as **catarrhal tonsillitis**, and presents the symptoms just described, the local signs being redness and swelling of the tonsils, which are often covered with viscid mucus.

A common form of inflammation of the tonsils is the **follicular** or **lacunar tonsillitis**. Here the inflamed crypts become filled with a cheesy material consisting of degenerate epithelium, leucocytes, sero-fibrinous exudate, bits of food, and countless bacteria. The tonsil, much reddened and swollen, is seen dotted here and there by these yellowish plugs peeping out from the mouths of the follicles, and from which the plugs can often be squeezed or scooped out *en masse*. There is commonly much pain in swallowing, rather high temperature, 102° to 104°, and marked general malaise. It is well to remember that the pain, both local and general, is usually less in diphtheria than in this form of disease, and that the fever of diphtheria, even in some of the severe cases, is often lower than that of lacunar tonsillitis.

PHLEGMONOUS TONSILLITIS.

Phlegmonous Tonsillitis, or quinsy, is often a recurrent affection. Sharp, darting pains running up toward the ear, pain on swallowing, perhaps a chill, announce to the patient the revisitation of the old disease. The pain becomes intense, swallowing and movement of the jaw are difficult, because of the pain, stiffness of the muscles, and swelling of the tonsils. The disease is usually, at first, unilateral. The tonsil is greatly swollen, dusky red, and covered with a pultaceous grayish exudate. When the amount of pus is large, fluctuation may be detected. Spontaneous or artificial evacuation of the abscess gives speedy relief. The pus will usually be discharged by the end of a week.

CHRONIC PHARYNGITIS.

Repeated attacks of acute pharyngitis, constant irritation from improper use of the voice, from the use of alcohol or tobacco, may result in a chronic inflammation of the pharynx. The patient complains of "hemming and hawking," of a tickling or rough feeling in the throat, and sometimes of pain. The throat is roughened from the distended mucous follicles, engorged and tortuous veins are seen coursing over the swollen mucous membrane, and there is usually a dusky red hue. Frequently this condition is combined with chronic inflammation of the tonsils and of the retro-nasal space.

In the atrophic form of pharyngitis the throat is smooth and shows a whitish or grayish color, from an overgrowth of connective tissue. From the lack of secretion, this form is often spoken of as **pharyngitis sicca.**

Chronic inflammation of the tonsils, or repeated acute attacks, often results in a permanent enlargement of the tonsils, an increase in the glandular structure, with an overgrowth of connective tissue, the latter making the tonsil not only larger than natural, but much firmer. The follicles are often distended with cheesy material, the patients are prone to be mouth-breathers, and are subject to repeated attacks of acute tonsillitis. The diagnosis is readily made by a physical examination of the tonsil.

It is not to be forgotten that the throat may be the seat of ulceration or inflammation of a syphilitic, tubercular, or malignant character, and that abscess, especially following an acute infectious disease, or tuberculosis of the vertebræ, may develop on the posterior pharyngeal wall. Recognition of the possibility of these occurrences will generally enable the diagnosis to be made.

DISEASES OF THE ŒSOPHAGUS.

ACUTE CATARRHAL ŒSOPHAGITIS.

This affection, usually of trifling clinical importance, may present such slight symptoms as to be unrecognizable. Odynphagia (pain on swallowing), and dysphagia (difficulty in swallowing), with occasional regurgitation of food, sometimes blood-stained, from a spasmodic stricture, may be present. A slight fever is occasionally met with. A knowledge of the operation of an efficient cause may lead to the suspicion of catarrhal œsophagitis. These causes are mechanical, as rough or sharp substances; chemical, as alcohol; thermal, as hot water. Extension of inflammation from the mouth and throat, or from the stomach, may produce the affection. During acute infectious diseases a mild grade of inflammation of the œsophageal mucosa often exists.

CHRONIC CATARRHAL ŒSOPHAGITIS.

Long continuance in operation of the causes of acute œsophagitis, chronic passive congestion of the mucous membrane, such as occurs in hepatic cirrhosis and in chronic respiratory and circulatory diseases, may lead to chronic œsophagitis. It is met with, too, in the vicinity of cancers and diverticula. The symptoms are seldom marked. Some pain or difficulty in swallowing may be noted, and mucus may be swallowed and then vomited, as though originally from the stomach.

PURULENT ŒSOPHAGITIS.

Localized or diffuse suppuration in the submucous tissue may be due to the spread of suppurative inflammation from neighboring organs, to the action of corrosives, or to arrested foreign bodies, as fish-bones.

Pain on swallowing, with more or less difficulty in swallowing, with the common symptoms of suppuration, pain, fever, at times chills and sweats, lead to the suspicion of the disease. The rupture of the abscess and the discharge *per os* or *per rectum* of pus, and the accompanying relief of symptoms may make the diagnosis more certain, though it is at times impossible to differentiate a genuine suppurative œsophagitis from a peri-œsophageal abscess with rupture into the œsophagus.

TOXIC OR CORROSIVE ŒSOPHAGITIS.

The history of the swallowing of some strong acid or alkali is obtainable in almost all cases. The odor of the breath or the color of the lips or skin about the mouth, the reaction to litmus of the inside of the cheeks, may give a clew to the poison taken. Thirst, great pain from the mouth to the stomach, often described as extending downward behind the sternum, vomiting, the vomited matter containing shreds and blood, prostration—these are the symptoms of this form of œsophagitis, some due to the severe stomatitis, pharyngitis, and gastritis which always accompany the œsophagitis.

It is important to remember that the prognosis in these cases must be influenced by a knowledge of the possibility of œsophageal stricture resulting from the involvement of the submucous tissue.

DILATATION OF THE ŒSOPHAGUS.

An enlargement of the œsophagus in its entire circumference is spoken of as a dilatation. It may be primary, from congenitally faulty muscular development, may be the result of a chronic catarrh, or, it is said, may follow a blow, lifting, the swallowing of very hot food. Secondary dilatation is found almost invariably above a stenosis. Dysphagia and regurgitation are the prominent symptoms. Pain is usually absent. The patient may learn to force food into the gullet with the muscles of the tongue, mouth, and pharynx, or may employ the fingers for this purpose. The regurgitated food, if it has not been immediately rejected, is alkaline, macerated, may show a trace of sugar, but none of the elements of the gastric juice. A large mass of food in the œsophagus may cause pressure-symptoms in the lungs or heart. The sound shows in the primary form a patent œsophagus, in the secondary an obstruction. Where there is dilatation, and at the same time a lengthening of the œsophagus so that it folds upon itself, the bougie may occasionally impinge against a fold of the œsophageal wall and give the impression of a permanent obstruction.

ŒSOPHAGEAL DIVERTICULA.

Diverticula are circumscribed dilatations or sacs. They are of two kinds—pressure-diverticula and traction-diverticula.

Pressure-diverticula, apparently due, in a majority of cases, to a congenitally weak musculature yielding to the continued intra œsophageal pressure of food, are met with oftenest in men over forty years of age. The most frequent site is the upper third of the œsophagus on the vertebral side. The disturbance in swallowing comes on gradually. Food "catches," stagnates in the diverticulum, decomposes, and imparts an offensive odor to the breath. The filled pouch may cause a swelling on the side of the neck which the patient may be able to empty by pressure. Clucking or gurgling sounds may be heard during the swallowing of food, or when the patient empties the sac. The decomposed food will occasionally be regurgitated. A large diverticulum may, when filled, cause pressure-symptoms, referable to the heart, trachea, bronchi, larynx, or, as it hangs down, it may act in reality as an extra-œsophageal cause of pressure-stenosis. The crucial test is the passage of the bougie, which at times enters the stomach unhindered, and at others catches in the diverticulum. The affection is rare.

Traction-diverticula, usually from adenitis in the neighborhood of the œsophagus, with resulting periadenitis, adhesions, and contraction, are oftenest met with near the bifurcation of the trachea in phthisical children. No diagnosis of the condition can be made. Perforation may cause communication between the œsophagus and a broken-down gland, a pulmonary cavity, the pleura, etc.

STENOSIS OF THE ŒSOPHAGUS.

The causes of œsophageal stenosis may be classified as follows:

1. **Intra-œsophageal**—foreign body, mass of food.
2. **Interstitial.**
   - *a.* Neoplasm.
   - *b.* Cicatricial tissue, as from toxic gastritis, ulcer (syphilitic, peptic).
   - *c.* Œsophageal abscess.
   - *d.* Diverticula filled—very rare.
   - *e.* Hypertrophy of musculature (?).
   - *f.* Congenital.
   - *g.* Spastic stricture.
3. **Extra-œsophageal.**
   - *a.* Enlarged glands, *e. g.*, sternal, cervical, bronchial, from tuberculosis, cancer, suppurative adenitis.
   - *b.* Mediastinal, or cervical cellulitis.
   - *c.* Vertebral disease.
   - *d.* Dislocation of hyoid bone or clavicle.
   - *e.* Cancer of pleura or lung.
   - *f.* Ossification of stylo-hyoid ligament, swelling of cricoid.

g. Pericardial effusion; cardiac hypertrophy; aneurism.
h. Dysphagia lusoria (?). Supposed to be due to an enlarged right subclavian artery, with anomalous course, crossing œsophagus.

The passage of the sound shows the impassability of the canal at the point of obstruction. English gum bougies of varying sizes, or the whalebone bougie, with olive-shaped tips, may be employed. But slight force is permissible. Aneurism, as an extra-œsophageal cause of obstruction, must always be excluded before passing a bougie, as, even in the hands of experienced clinicians, the aneurismal sac adherent to the œsophageal wall has been ruptured, with fatal result. The calibre of the stricture is to be noted, the number of strictures, for there can be several, and the distance from the incisor teeth. The cardia, in an adult of average height, is sixteen inches from the incisor teeth, measured by an œsophageal sound. Where stenosis is complete there is no deglutition-sound upon auscultation.

Unless there is a suddenly operating cause, there is, first a complaint of difficulty in swallowing solids, then semi-solids, and, finally, even liquids. The point beyond which the food cannot pass is sometimes fairly well located by the patient. In the early period he will often attempt to swallow by the aid of water. The food accumulates in the œsophagus, which commonly dilates above the point of stenosis, becomes macerated, the salivary amylolytic action may continue until, finally, there is regurgitated the softened, perhaps decomposed food, non-digested, and often giving the reaction for sugar. The effect upon the general health depends both upon the condition producing the stenosis and upon the extent to which starvation is caused by stenosis. A mass of food accumulated above a stenosis may, by pressure, cause anxiety, intra-thoracic pain, or dyspnœa.

Spasmodic stricture may be suspected when we have inability to swallow in an hysterical, hypochondriacal, or markedly neurotic patient, or when it occurs during rabies, or tetanus, or after the ingestion of a large amount of belladonna. There is, in this variety of œsophageal stricture, often a history of the transitory character of the dysphagia, and the sound, if necessary passed during anæsthesia or morphine-narcosis, clears up the diagnosis.

CANCER OF THE ŒSOPHAGUS.

This is generally primary, found oftenest in the lowest third, insular or ring shaped, preventing the passage of food, by projecting into and blocking up the lumen, or by its growth and richness in connective tissue immobilizing the muscular tissue, all peristalsis being thus checked. Neighboring lymph-glands are often carcinomatous; ulceration with hemorrhage, sloughing, or perforation may occur. Adult males are oftenest affected. An œsophagus previously injured by trauma, foreign bodies, chronic catarrh, is said to be predisposed to carcinosis.

The symptoms are those of œsophageal stenosis, with the constitutional manifestations of cancer. The symptoms of obstruction are gradually more and more manifest; pain may be insignificant. When dilatation has taken place above the obstructing growth food is retained for some time and then regurgitated—vomited, the patient often thinks. To the evidences of starvation are added those of the cancerous cachexia, with its accompanying anæmia. Slight fever is not uncommon. Indican is often present in the urine. Secondary glandular or visceral deposits may be found. The sound detects the obstruction, often, unless cautiously employed, causing a little bleeding. Fragments of the new growth, brought up in the eye of a stomach-tube, may furnish microscopic evidence of the cause of the stenosis. Death occurs usually inside of two years from (*a*) stenosis and starvation; (*b*) marasmus, where stenosis is not complete; (*c*) hemorrhage; (*d*) perforation of œsophagus; (*e*) invasion of some other organ, as stomach, spinal cord; (*f*) complications, as pulmonary phthisis.

The diagnosis is usually made with little difficulty, by considering the age, sex, cachexia, secondary deposits, fragments of tumor, stenosis, exclusion of other causes of stenosis.

PERFORATION OF THE ŒSOPHAGUS.

Perforation may be caused by wounds external or internal, by corrosives, by ulceration such as occurs in carcinoma, or by extra-œsophageal causes, as the rupture of a para-œsophageal abscess or aneurism.

Pain and nausea, with collapse, mark the accident. The subsequent symptoms depend on the organ or space communicated with. Thus mediastinal or cervical cellutitis, suppurative pleurisy or pericarditis may be sequelæ. If a large vessel is opened, fatal hemorrhage ensues; if a bronchus, there is coughed up food or liquid when the attempt is made to swallow, and colored liquid or milk has been given in these cases as an aid to diagnosis; and bronchitis, pneumonia, or gangrene usually follows.

Spontaneous Rupture has occurred, especially in male alcoholics, where no trauma or previously existing disease could be proven. The sudden pain, sense of something bursting, the vomiting, collapse, evidence of air and fluid in the pleural cavity, subcutaneous emphysema, may give grounds for a diagnosis of this rare affection. Death usually occurs inside of twenty-four hours.

Œsophagomalacia cannot be diagnosed unless perforation occurs where it can only be suspected. Some deny an antemortem softening of the œsophageal wall.

PARALYSIS OF THE ŒSOPHAGUS.

This has been met with as a part of the symptom-complex of brain and spinal cord disease, and in consequence of diphtheria and syphilis. The knowledge of the existence of an efficient cause, the inability to swallow, the perviousness of the œsophagus to the sound, enable us to make the diagnosis.

HEMORRHAGE FROM THE ŒSOPHAGUS.

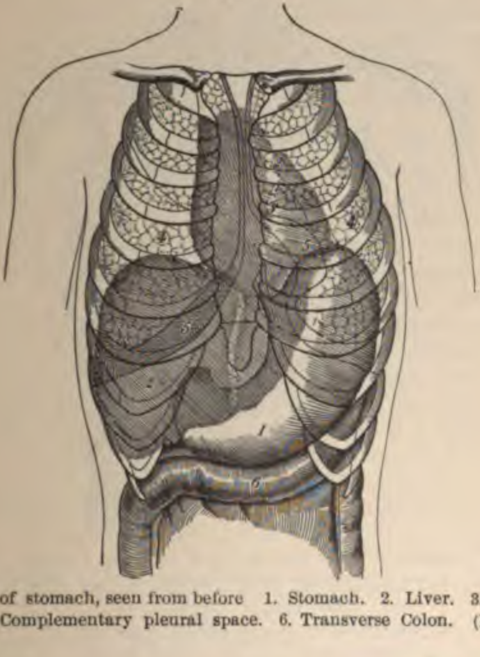
Hemorrhage from œsophageal bloodvessels can only be recognized as such by excluding hemorrhage from the mouth, nares, pharynx, stomach, and intestines. Blood from the œsophagus usually passes into the stomach and is vomited or passed at stool. Wounds, ulcers of various kinds, rupture and perforation of the œsophagus may cause hemorrhage. The lower œsophageal veins, by virtue of their anastomoses with the gastric veins, may become varicose because of portal obstruction, as in cirrhosis of the liver. Several cases of fatal hemorrhage from rupture of varicose œsophageal veins are reported.

DISEASES OF THE STOMACH.

GENERAL CONSIDERATIONS.

The following facts concerning the anatomical position of the stomach are worth special consideration. Four-fifths of the organ lie to the left of the median line, the pyloric one-fifth to the right. The upper limit of the stomach reaches the fifth rib or even the fourth interspace; the lower limit of the greater curvature does not reach as low as to the umbilicus. The cardiac opening, which is opposite the junction of the sixth or seventh costal cartilage with the sternum, the lesser curvature, pylorus, are beneath the overlapping edge of the liver. A stomach with a capacity of more than 1600 c.cm. is regarded as abnormally enlarged.

FIG. 17.



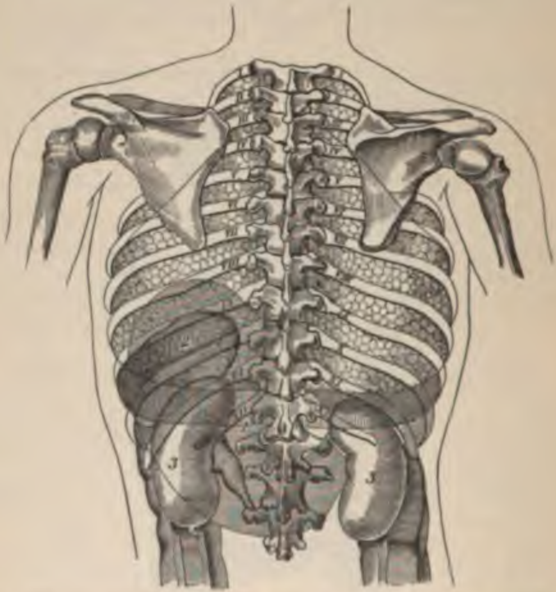
Position of stomach, seen from before 1. Stomach. 2. Liver. 3. Heart. 4. Lungs. 5. Complementary pleural space. 6. Transverse Colon. (EICHHORST-BOAS.)

METHOD OF DETERMINING THE POSITION AND SIZE OF THE STOMACH.

Inspection, palpation, percussion, auscultation, are here employed as in the case of thoracic diseases. In order to aid the physical examination various agents have been employed to distend the viscus, so that its size and position may be more readily appreciated.

(*a*) Air injected by means of a stomach-tube and an ordinary gas-bag can be used for purposes of inflation. This is better than the following method, as the amount injected can be regulated and the stomach can be alternately filled and emptied until the examiner is satisfied as to the results of his examination. One drawback is the liability of the escape of air by the side of the tube.

FIG. 18.



Position of the stomach, seen from behind. 1. Stomach. 2. Spleen. 3. Kidneys. (EICHHORST-BOAS.)

Instead of the gas-bag it is easier and better to employ a bulb such as accompanies a Paquelin cautery. In this way air can be carefully and steadily forced into the stomach. There is, however, necessity for great caution for fear of overdistention. I have seen post mortem, several linear tears extending through the mucosa and into the muscularis in a stomach inflated with air five days before, in one of the best clinics of Europe.

(*b*) A teaspoonful each of bicarbonate of soda and tartaric acid dissolved separately in a small amount of water and drank, the one after the other, inflates the stomach with CO_2. Severe pain, even collapse, is sometimes caused; there is some danger of suddenly overdistending the organ, and we cannot regulate accurately the amount of gas in the stomach. Still, this is a simple method and one requiring no apparatus.

(c) Water swallowed or passed through a stomach-tube causes an alteration in the percussion-line of dulness, the patient being examined in the erect position. In many cases this is the simplest and surest method of diagnosis. It is known as Penzoldt's method. Where the abdominal walls were thin and cold water has been employed I have been able, by palpation, to locate the greater curvature with considerable accuracy by noting the difference in temperature as felt by the hand. The colon has also been distended by gas or water, in order to make the differentiation between its limits and those of the stomach easier.

Gastrodiaphany, the lighting up of the stomach by means of an electric light passed into the stomach, has, perhaps, a limited use in determining the size and outlines of the organ and the presence of growths on the anterior wall.

Inspection seeks for prominences and irregularities of contour, and takes note of increased vermicular motion. *Percussion* is often of limited value, because of difficulty of distinguishing the resonance of the neighboring lungs and intestines from that of the stomach. By auscultation over the xiphoid cartilage a first murmur is heard at the time of swallowing. A second murmur, heard about twelve seconds later, is, in obstruction of the cardia, delayed and changed in timbre. When gas and fluid are in the stomach the *succussion*, or splashing, sound is elicited by shaking the patient. This is especially noticeable in dilatation. *Palpation* is, in the majority of cases, of more value than any other method of examination. Points of tenderness or induration, vermicular movements, and in thin patients with hypertrophied stomach walls, even the limits of the organ may be found by palpation.

The contents of the stomach may be obtained for examination by a stomach pump, or catheter. By the latter method, the simpler and safer, a soft-rubber stomach-tube, preferably with a terminal and one or two lateral openings, is passed into the stomach, and the patient, by contraction of the abdominal muscles, perhaps aided by pressure of the physician's hand, forces out the contents—produces an artificial emesis. At times it is necessary, in order to start the flow, to empty the tube of air by "stripping it." Where there has been hemorrhage from the stomach, or there is suspicion of ulcer, great caution is necessary, to avoid starting a second hemorrhage. Ewald's advice is against the use of the stomach-tube in ulcer, unless absolutely demanded.

Test meals. Leube's meal consists of a few slices of bread, a plate of soup, and a piece of beefsteak. At the end of seven hours all traces of food in the stomach should be absent. Ewald's test-breakfast, of a cup of water, or weak tea, with a roll or biscuit, given after a fast of twelve hours, is the simplest and best. One hour after its administration the stomach should show free hydrochloric acid, pepsin, milk-curdling ferment. Organic acids should be absent, or present only in the minutest traces.

The following scheme is modified from Boas, and gives in outline the method of examination of stomach-contents, which are to be filtered:

1. Examination with the naked eye, there being noted (*a*) the appearance, (*b*) the quantity, (*c*) abnormal substances, as blood, bile, pus, etc.

2. Chemical examination of the filtrate.

(*a*) Reaction with litmus-paper.

(*b*) Reaction with Congo-paper. Red color turned to blue by free acids.

(*c*) Examination for free hydrochloric acid.

The following solution is necessary: Phloroglucin 2, vanillin 1, absolute alcohol 30. A few drops of this solution and of the stomach-fluid (equal parts) gradually heated to dryness on a white porcelain dish, show a beautiful scarlet or rose red color, showing first at the edges, if free HCl is present. In place of this—Günzburg's test solution—that of Boas may be employed, possessing the advantages of stability and cheapness. Boas' solution is made of resorcin resub. 5, sacch. alb. 3, spirit dil. 92. The reaction is the same as with the Günzburg solution. Approximate quantitative estimation of free hydrochloric acid can be made by noting the intensity of the color, with either of these tests, just as we are able with litmus-paper to say that a solution is strongly or faintly acid, according to the intensity of the red color given to the litmus-paper.

(*d*) Examination for lactic acid by Uffelmann's method, or, in doubtful cases, the shaking up of the filtrate with ether, and examination of the sediment after ether-evaporation and addition of a little water. Uffelmann's test is as follows: To 20 c.cm. of a 5 per cent. solution of carbolic acid add 2 or 3 drops of the tincture of the chloride of iron, and then water until an amethyst-blue color is produced. A few drops of a solution of lactic acid will change 1 c.cm. of this iron solution to a lemon-yellow tint.

(*e*) If suspicion of butyric or acetic acids, test by ether-method.

(*f*) Examination for pepsin. A shaving of coagulated egg-albumen is kept in the filtrate one hour at the body-temperature. If this disappears in the presence of free hydrochloric acid, pepsin is present.

(*g*) Examination for milk-curdling ferment. This is proven by the coagulation of neutral milk by neutralized stomach-contents. Practically, the presence of free HCl. has been found to imply the presence also of pepsin and of rennet-ferment.

(*h*) Quantitative estimation, where desired, of free and combined acids.

(*i*) Examination of power of digestion of albuminoids and carbohydrates.

In the majority of cases the examination need not extend beyond (*d*).

3. **Microscopic Examination** for (*a*) starch, flesh, fat-globules, etc., (*b*) abnormal substances—*e. g.*, epithelium, blood, leucocytes, bacteria, fungi, etc.

The **propulsive power** of the stomach and patency of the pylorus are best determined by Ewald's salol-test, though it is not fully satisfactory. Fifteen grains of salol are given and the urine examined for the first appearance in it of salicyluric acid, shown by the violet color on addition of a few drops or a solution of ferric chloride. As the salol must have entered the alkaline intestine before being split up into carbolic and salicylic acids, this test shows motor efficiency of the stomach. Normally, the urine shows the reaction in one hour.

The **absorptive power** is proven by giving in a capsule, the outside of which has not been touched by the drug, a few grains of potassium iodide. When absorption is normal, in from three to twelve minutes, the saliva shows, on the addition of starch and nitric acid, the blue reaction. The test should be made with the stomach empty.

The easy recognition, trivial character, and self-limitation of many gastric disorders make the use of the stomach-tube in these cases unneccessary, especially in private practice, where politic reasons also may, at times, render its employment unwise. In many conditions, however, it is the only sure means of arriving at a scientific and accurate diagnosis, and a consequent rational therapy. It permits of early diagnosis before physical signs are marked enough to be of aid; it confirms the statements of patients and the examination of the physician in doubtful cases; while in many instances it shows that the complaints of patients, as to food remaining in the stomach long after eating, sour stomach, etc., are contradicted by the facts, and that the patient is a sufferer from nervous disarrangement. The practical value of examination of stomach-contents will be seen as separate diseases are considered.

GASTRITIS.

Acute Catarrhal Gastritis.

In this form, as in all others, a knowledge of the existence of a causal factor is of the greatest importance as regards diagnosis. Thermic, mechanical, chemical causes may induce a primary acute gastric catarrh. Thus, too hot or too cold food, exposure to cold, the swallowing of rough substances, of large masses of indigestible food, may be causes; and under the head of chemical agents must be classed many articles of poorly cooked foods, spoiled meats, etc., as well as alcohol and the chemical products of bacterial action. Secondary catarrhal inflammation of the stomach occurs during many of the acute infectious diseases, and by extension of inflammation from the intestine or œsophagus.

Pain and tenderness in the epigastrium with loss of appetite call attention to the stomach. There are often nausea and vomiting which may, by getting rid of the offending material, be followed by speedy recovery. Headache is common as well as malaise—both evidences of auto-intoxication. The temperature and pulse are a little above normal. The tongue is coated, there is a bad taste in the mouth and often an offensive breath. Bowel-disturbances are common.

In some of the severer cases, as after taking large amounts of tainted fish, vomiting and purging, with prostration, may be extreme.

At times the disorder is of a subacute character attended by a continued fever, great gastric irritability, and many appearances analogous to typhoid fever. Such cases are sometimes spoken of as gastric fever. They may last for several days or weeks.

Acute Toxic or Corrosive Gastritis.

Acute toxic or corrosive gastritis is caused by the ingestion of some of the strong poisons, as the mineral acids, the alkalies, corrosive sublimate, etc. The history of the accident or attempted suicide usually enables us to make a diagnosis, or the discovery of the empty bottle puts us on the right track. The very sudden onset of this form of disease, the great pain, uncontrollable vomiting, often of blood and shreds of mucous membrane, the intense thirst, great prostration, purging, make the diagnosis clear. The odor of the breath may enable one to tell the nature of the poison, as may the appearance of the mouth and lips. Thus nitric acid causes the familiar yellow stain on the skin, strong sulphuric acid chars the skin, carbolic acid whitens it. The reaction of the mucous membrane of the mouth, as shown by litmus-paper, will sometimes enable one to decide whether an acid or an alkali has been swallowed.

Acute Suppurative or Phlegmonous Gastritis.

Acute suppurative or phlegmonous gastritis is a rare disease, often secondary to suppurative disease in some neighboring organ, or a part of a general septic process, the knowledge of which primary disease is usually our only clew to the nature of the gastric disorder. The symptoms are much the same as those of acute catarrhal gastritis save that there are greater pain, often more localized, higher fever, more marked prostration, and often chills, and sweats. Rupture of the abscess into the stomach is followed by the appearance of blood and pus in the vomitus or feces. A positive ante-mortem diagnosis of this disease is rarely made.

Chronic Gastritis.

Long-continued errors in diet, the use of alcohol, repeated attacks of acute gastritis lead to primary chronic gastritis. Secondarily the stomach may be involved in diseases attended by portal obstruction, in gout, rheumatism, diabetes, Bright's disease. A chronic catarrhal inflammation usually accompanies carcinoma and ulcer of the stomach.

Gastric juice inferior in quality, deficient in hydrochloric acid, fails to digest the food as rapidly as it should, and permits fermentation—*e. g.*, lactic-acid fermentation—to go on. We find, therefore, patients complaining of a sense of weight in the stomach long after meals and of pain more or less severe.

The gases formed may cause distention or eructations. A feeling of oppression of breathing or of palpitation may be present. Mucus, poured out in increased quantities, wraps itself around food-particles, preventing the gastric juice from coming in intimate contact with the food, and also, by its alkalinity, neutralizing the acidity of the stomach-contents. Nausea and vomiting may be marked. Acute exacerbations of chronic gastritis may follow excessive or improper indulgence at the table. The tendency is for these cases to grow worse as there is more and more glandular structure annihilated, and especially if it happens, as it often does, that the stomach-wall gradually weakens, gives way, and a condition of true dilatation occurs. The motor and absorptive power of the organ, as well as its digestive function, being interfered with, it is not surprising that evidences of malnutrition are marked in some of these cases, and especially where auto-intoxication by absorption from the stomach of the abnormally formed chemical substances is excessive. Constipation often adds to the difficulties. These patients are, therefore, often emaciated, of a muddy color, languid, sufferers from nervous irritability, even moroseness and hypochondriasis. Headache is common, the appetite is capricious, the tongue coated, breath offensive, sleep often restless. Secondary functional derangement of other organs often leads patients to think the trouble is in the brain, heart, kidneys.

Free hydrochloric acid is usually in lessened quantity, or absent; organic acids are frequently present, digestion is slow, the salol-test reveals deficient propulsive power, and the iodide-test a slow rate of absorption.

If complete atrophy of the glandular structure occurs, not only will HCl, but pepsin and the milk-ferment be absent. A gradually progressive anæmia follows.

(For differential diagnosis between ulcer, cancer, and chronic gastritis, *vide* p. 102.)

PEPTIC ULCER OF THE STOMACH.

The impairment of circulation over a limited area of the stomach-wall, as by the blocking of a terminal vessel by an embolus or thrombus, brings the resisting power of this tissue, now poorly nourished, so much below par that it must submit to digestion by the gastric juice. And this is especially true if the blood is impoverished, as in anæmia and chlorosis, and the gastric juice rich in hydrochloric acid. The result of this digestion of the stomach-wall is the round or peptic ulcer, not, from the standpoint of pathological anatomy, an ulcer so much as a "progressive necrosis of tissue," as Virchow terms it.

Considering its etiology, we are not surprised, therefore, to find it oftenest in sufferers from anæmia and chlorosis, girls of from fifteen to thirty, and those weakened by infectious diseases. Alcoholics, gourmands, sufferers from arterio-sclerosis are named, as often affected. It may occur in connection with extensive burns of the surface of the body.

The diagnosis is made from a consideration of the above predisposing factors, and upon the existence of pain, hemorrhage, vomiting, excess of hydrochloric acid.

The typical **pain** is localized, worse after eating. In other cases it is irregular as to time of occurrence and location, resembling the pain of gastralgia. It may be boring, lancinating, or aching in character.

Vomiting is common, particularly after meals, and usually affords relief from the excruciating pain.

Hemorrhage is present in fully one-half the cases. The blood appears either in the stools or, oftener, in the vomitus. Small amounts may be vomited at times, or a sudden hæmatemesis may be followed by syncope, profound or even fatal anæmia. The sudden vomiting of a large amount of arterial blood is quite characteristic of ulcer. Sudden and fatal hemorrhage may occur before blood is vomited or passed at stool. "In ulcer of the stomach the gastric juice always contains hydrochloric acid, and usually an excess of it." (Ewald.) The digestive, motor, and absorptive functions are rapidly performed Digestion of the starches is, however, owing to the hyperacidity, retarded.

With this group of classical symptoms the diagnosis is easy. In cases, however, pain may be trifling, and a sudden severe hemorrhage or a perforation-peritonitis be the first evidence of disease.

When there is much vomiting the general health may suffer considerably, though in cases the patient's healthy appearance is in strange contrast to the serious complaint. The bowels are often constipated, the urine scanty and of high specific gravity. The appetite may be good, the tongue is often clean and red. There is rarely a slight fever. Recovery after weeks, months, or years, is common, yet it is to be remembered that recidivation is frequent, painful cicatrices may be a source of annoyance or of serious deformity of the stomach, and carcinoma may develop on the site of a previous ulcer.

CANCER OF THE STOMACH.

Carcinoma ventriculi is usually primary, most frequently found in males between forty and seventy. Heredity seems to have some influence as a predisposing factor. While oftenest located at the pylorus or cardia, it may be situated in any portion of the stomach-wall.

The first symptoms are those of chronic gastritis, anorexia, distress after eating, flatulence, occasionally nausea and vomiting. Pain may be absent or marked. There is a slow but progressive loss of strength and of flesh, and this may be noticed even before gastric symptoms appear. Anæmia increases. Constipation is the rule, the urine is scanty, often containing indican. Vomiting becomes common, the vomitus containing blood in small quantities, changed by the action of the gastric juice to a dark "coffee-ground" looking material. The vomiting, where secondary dilatation occurs, will take place characteristically, at long intervals, but be of large amount. The patient may survive several months, or even two or three years from the time of beginning of the new growth. Death may occur from marasmus, starvation, hemorrhage, perforation, intercurrent disease, or, by secondary involvement of other organs, from general carcinosis.

When we meet in a male over forty, with these symptoms of indigestion with no dietary cause, progressive emaciation and weakness, development of cachexia, vomiting, often with coffee-ground material in the vomitus, pain in the epigastrium, there is *prima facie* evidence of cancer of the stomach. An examination of the epigastrium will show, in the majority of cases of carcinoma, a painful mass noticeable on inspection or palpation. There is often a dislocation of the pylorus, so that the induration may be felt even as low down as to the umbilicus. It may be freely movable. In some cases, where the tumor is at the fundus or on the posterior wall, the diagnosis, because of lack of vomiting and failure to detect a mass, is a matter of great difficulty or is even impossible. Pain is sometimes curiously absent.

Gastrectasia is common, and proof of its existence is of great importance in doubtful cases. Often early, and, with rare exceptions, always late, in the disease free hydrochloric acid is absent. The abdominal and inguinal lymph-glands may be enlarged, and especially, it is said, the clavicular glands of the left side. The exaggerated peristaltic movements of the stomach are at times plainly visible through the thinned abdominal wall.

Recently greater stress has been laid upon the detection of lactic acid after test-meals than upon the failure to find free hydrochloric acid, as an early evidence of carcinoma ventriculi. As offering less possibility for the introduction of lactic acid into the stomach through baked foods, Boas recommends a meal of a thin gruel made of a tablespoonful of oatmeal-flour to the litre of water, a little salt being added to make it palatable. This is to be taken at night after gastric lavage. In the morning the stomach-contents are obtained in the usual manner and tested by Uffelmann's method for free lactic acid. Boas,[1] Stewart, and others are of the opinion that only in carcinoma is there sufficient lactic acid to give under these conditions the Uffelmann reaction—*i. e.*, the canary-yellow tint to the carbolic-ferric-chloride solution.

It may be well to call attention to a very common mistake that is often made regarding the diagnostic value of the absence of free HCl from the stomach. Absence of free HCl does not of necessity indicate carcinoma. In cases of gastrectasis, of chronic gastritis, in some cases of functional gastric disease, there may be absence of the free acid for days or weeks, so that the absence of free HCl is not a positive indication of carcinoma. Taken in conjunction with other symptoms and findings, it is a confirmatory evidence. In a suspected case, however, the finding of free HCl tends to exclude carcinoma, as it is rare, except early in the disease, to find the free acid. And yet even the finding of free HCl does not positively exclude carcinoma, as in cases where the neoplasm has developed in the cicatrix of a round ulcer free acid has been found even quite late in the course of the disease. Unless the lactic-acid test is infallible, one must still rely for the diagnosis of carcinoma upon the complexus of symptoms and not upon any one.

[1] Münchener med. Wochenschrift, Oct. 24, 1893.

A more accurate method for testing for lactic acid is described by Boas in Deutsche med. Wochenschrift, Sept. 28, 1893.

DIFFERENTIAL DIAGNOSIS OF CANCER, ULCER, AND CHRONIC GASTRITIS.

Cancer of the stomach.	Ulcer of the stomach.	Chronic catarrhal gastritis.
1. Usually over age of forty years.	Usually fifteen to forty years of age.	At any age.
2. Cause unknown. In cases heredity.	Cause unknown. Often in chlorotic. May be history of previous attacks.	Usually errors in diet, alcoholic excess, or some primary chronic disease, as nephritis, tuberculosis, hepatic cirrhosis, valvular heart-disease.
3. Rarely lasts longer than two years. Always fatal.	May last from few weeks to several years. Relapses common.	Of indefinite duration. Amelioration or recovery under treatment. Relapse common.
4. Tumor in most cases (80 per ct.). Secondary cancers at times found in liver, peritoneum, lymph-glands.	Tumor seldom present (hypertrophy). No secondary tumors.	Primary and secondary tumors absent.
5. Appetite poor. Often loathing of meat. Tongue pale, with furry coat.	Appetite between attacks of pain good. Tongue usually clear, red.	Appetite capricious. Tongue usually coated.
6. Vomiting common. At short intervals or, when dilatation exists, at long intervals and of large amount. Rarely cancerous fragments found.	Vomiting not so frequent as in cancer, and never, except rarely late in the disease, characteristic of dilatation.	Vomiting often present only at times of exacerbations of the gastritis. May be absent for weeks at a time.
7. Hemorrhage frequent; rarely profuse; often gives coffee-grounds appearance to vomitus.	Hemorrhage less frequent than in cancer, but more profuse.	Hemorrhage rare.
8. Free hydrochloric acid usually absent from stomach-contents, at least late in the disease. Lactic acid said to be present (Boas.)	Free hydrochloric acid always present and usually in excess.	Free hydrochloric acid may be present or absent. Organic acids often present.
9. Digestion usually slow and imperfect.	Digestion of starches retarded, of meat rapid.	Digestion often imperfect.
10. Pain less dependent than in ulcer upon presence of food in stomach, less localized, more continuous.	Pain often paroxysmal, localized, worse after eating; relieved by vomiting.	Pain usually less severe than in cancer or ulcer; more irregular in its manifestations.
11. Emaciation, loss of strength, cachexia, anæmia, often out of proportion to gastric symptoms. Leucocytosis.	Cachexia, weakness, etc., often absent; when present, in proportion to severity of gastric symptoms, vomiting, hemorrhage, pain.	No cachexia unless gastritis is secondary to some disease, as tuberculosis, nephritis, etc.
12. Perforation rare.	Perforation commoner than in cancer.	Perforation does not occur.

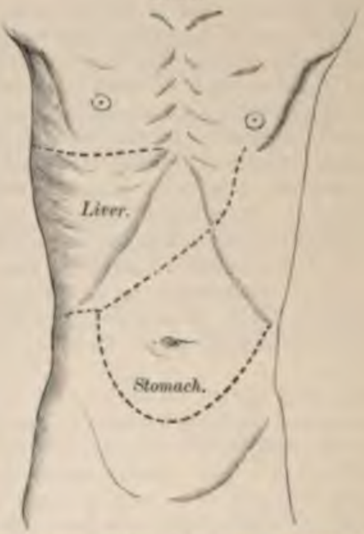
DILATATION OF THE STOMACH.

Clinically, a dilated stomach is one that is not alone anatomically enlarged but physiologically imperfect, functionally inefficient, chiefly as regards its motor or propulsive power. The most common cause is stenosis of the pylorus or duodenum, producing hypertrophy of the musculature with gastrectasia. Neoplasms and, next in order of frequency, cicatricial tissue furnish the commonest causes of obstruction. Pressure from without, as from an aneurism or new growth, may result in obstruction. More rarely muscular hypertrophy, torsion of the duodenum, are the causes. The stenosis may possibly have been congenital in a few reported cases. In another group of cases dilatation is caused by an excessive amount of ingesta, as in gourmands, especially if the food be indigestible or poorly masticated. Chemical insufficiency also with stagnation and fermentation, gradually leads to muscular insufficiency and dilatation. In a third group the muscular force is impaired through (1) organic changes, as in ulcer, carcinoma, inflammations, and degenerations, such as fatty or amyloid; (2) through mechanical restraint, as where adhesions exist or the stomach-wall is fixed by the dragging of a hernia; (3) through impaired nutrition, as in anæmia, typhoid, tuberculosis.

In attempting to diagnose dilatation the symptoms of the primary disease must be kept clearly in mind—*e. g.*, carcinoma, cicatrix following ulcer, anæmia, chronic gastritis with chemical insufficiency. The characteristic symptom is the vomiting at long intervals of a large amount of partially digested food, some of which may have been taken one or two days before. Patients are often astonished to see that a few hours after a meal they eject more food than was taken at the meal just completed. "They vomit more than they eat." If stenosis is complete, there is no bile in the vomitus. Symptoms of chronic gastritis, anorexia, pyrosis, eructation, fulness or pain, with impairment of general health and often marked nervous debility, are common. Thirst, palpitation, constipation, are frequently noted.

Inspection will at times show the enlarged outline of the stomach, especially if it be filled with food, water, or gas (*vide* p. 92). Peristalsis is often visible. Palpation often determines the location of the greater curvature, and a splashing sound is elicited by sharp percussion. A stiff stomach-tube or bougie passed into the stomach as far as safely possible can be felt below the umbilicus (Leube). Some danger attends this rather uncertain method of diagnosis, and Leube himself has practically discarded it. The bubbling or sizzling sound of fermentation is sometimes heard with the stethoscope. Chemically, HCl is diminished or absent, organic acids present. Leube's meal remains undigested at the end of seven hours. Absorption and propulsion are shown to be slow by the iodide and salol tests.

FIG. 19.



Limits of a dilated stomach distended with gas, as obtained by percussion. (EICHHORST.)

Aufrecht[1] has recently described another confirmatory test of dilatation. Percussion of some portion of the dilated viscus will reveal slight dulness, while tympany is noted elsewhere. If a few seconds later this same spot be percussed, tympany will be found where there was before dulness. This changing percussion-note, due probably to the contraction of the thickened wall during peristalsis, he regards as diagnostic of dilatation. Associated with this is often a sound similar to the cracked-pot sound as heard over pulmonary cavities.

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[1] Centralblatt für klin. Med., 1893, No. 23.

HEMORRHAGE FROM THE STOMACH.

The following classification includes the causes of hemorrhage from the stomach, a knowledge of which causes is of great aid in diagnosis:

1. **Trauma.** (*a*) *External,* as from bullet, knife, fractured rib perforating stomach-wall, contusion causing rupture.

(*b*) *Internal,* mechanical, as roughly handled sound or stomach-pump; chemical, as acids, alkalies; thermic, as boiling water.

2. **Primary Stomach Disease**—*i. e.*, **Local Causes.**

(*a*) Peptic ulcer; rarely tubercular ulcer.

(*b*) Carcinoma.

(*c*) Phlegmonous, acute, and chronic catarrhal gastritis.

(*d*) Varicose veins and aneurisms of gastric vessels.

3. **Remote Causes**—*i. e.*, **Primary Disease Elsewhere than in Stomach.**

(*a*) Congestion of portal system from hepatic disease, as cirrhosis or carcinoma; pulmonary disease, as emphysema; cardiac disease, as dilatation or uncompensated valvular diseases.

(*b*) Infectious diseases. Yellow fever, variola, malaria, scarlatina.

(*c*) General diseases. Hæmophilia, scurvy, progressive anæmia.

(*d*) Uræmia, cholæmia; hysteria, vicarious menstruation.

(*e*) Perforation of stomach-wall from bursting of aneurism or abscess.

Trifling hemorrhage produces no symptoms. Severe hemorrhage may result in sudden, fatal collapse without the appearance of blood externally. Usually there is the vomiting of blood—hæmatemesis, or the appearance of blood in the stools. It is well to remember that blood from the stomach passed at stool is dark and fetid. The nearer the site of the hemorrhage to the anus the brighter and fresher the blood in the stools. The symptoms and signs of acute anæmia follow a gastric hemorrhage of any considerable extent. Dizziness, tinnitus, yawning, sense of faintness or even syncope, rapid pulse, pallor are met with as in acute anæmia from any other cause. A sense of heat and weight in the epigastrium may be complained of.

The diagnosis rests, therefore, upon a knowledge of the existence of a disease capable of producing gastrorrhagia, the symptoms and signs of acute anæmia, the vomiting of blood or passing it at stool. So important an element in diagnosis is hæmatemesis, and so easy is it at times to confuse it with hæmoptysis, that the following scheme of differential diagnosis, very slightly modified from Welch,[1] is appended.

HÆMOPTYSIS.	HÆMATEMESIS.
1. Usually preceded by symptoms of pulmonary, bronchial, or cardiac disease.	1. Usually preceded by symptoms of gastric or hepatic disease or other diseases mentioned under etiology.
2. The attack begins with a tickling in the throat or behind the sternum. The blood is coughed up. If vomiting occurs, it follows the cough.	2. The attack begins with a feeling of heat and fulness in the stomach, followed by nausea, and the expulsion of blood by vomiting. If cough occurs, it is secondary,
3. The blood is bright red, frothy from admixture of air, alkaline; is seldom coagulated, and often contains muco-pus.	3. The blood is dark, often acid, usually mingled with food and other contents of the stomach.
Blood that has remained long in the bronchi or a cavity, is dark and coagulated.	If the blood is vomited immediately after its effusion, it is bright red and alkaline. If effused into an empty stomach, it may be alkaline.
4. The attack is usually accompanied and followed by localized moist râles in the chest, and there may be, besides, the physical signs of pulmonary, cardiac, or bronchial disease.	4. Physical examination of the lungs after the attack is usually negative, but there are generally symptoms and signs of gastric or hepatic disease.
5. Bloody sputum continues often for several days after the hemorrhage.	5. Black stools usually follow hæmatemesis.

When blood is vomited we must exclude hemorrhage from the mouth, throat, nose, or œsophagus, and subsequent swallowing of the blood, before pronouncing the case one of gastric hemorrhage. So, too, it is to be remembered that malingerers may swallow blood for purposes of deception, and that the juices of some berries and medicinal agents may by their color mislead both the patient and physician. It is rarely necessary to employ the microscope or the test for hæmin crystals.

Following a gastric hemorrhage we may have the anæmic symptoms—pallor, weakness, œdema, amaurosis, albuminuria, alopecia. Until the blood is entirely out of the stomach and bowel a slight rise in temperature may be observed.

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[1] Pepper's System of Medicine, vol. ii. p. 585

FUNCTIONAL DISEASES OF THE STOMACH.—NEUROSES.

In this class of cases no anatomical lesion is found to account for the symptoms, which appear to be due to perverted nerve-action. As the nerves of the stomach are motor, sensory, and secretory, the functional disturbances, naturally, fall into three corresponding groups, *motor, sensory*, and *secretory* neuroses. Clinically, the sharp line of distinction between these different groups cannot always be accurately drawn, many cases being complex in character, so that a *mixed* group is formed.

MOTOR NEUROSES.

(*a*) Nervous Vomiting.

Nervous vomiting is vomiting where no alteration of the stomach or its contents is discoverable, but where, through irritation of the vomiting-centre, often from causes operating at a long distance, emesis is induced.

This form of vomiting is often met with in hysterical women. Under this group may be classed the reflex vomiting of cerebral disease, of renal colic, intestinal, uterine, or ovarian diseases, tabes dorsalis, uræmia, etc. Preceding the ejection of the stomach-contents there is not the depressing nausea, nor the straining and retching of ordinary vomiting. The act more nearly resembles regurgitation. Symptoms and signs of gastric disease are lacking. There is often a persistently clean tongue.

(*b*) Nervous Eructation.

Eructation of gas, often but a symptom of a gastric disorder, at times, in neurasthenic and hysterical patients, becomes the predominating feature of the case and rises to the dignity of a disease. The gas ejected is odorless, tasteless, apparently identical with air. Eructation may occur periodically or be irregular. A case is recorded in which during an attack there were, in the course of an hour, 2500 eructative explosions.

(*c*) Rumination.

There are several well-authenticated cases on record where food, after being swallowed, has been regurgitated and rechewed, after the manner of the cud-chewing animals. The affection is found oftenest in idiots, hysterical patients, or those of a decidedly neurotic temperament.

(*d*) Insufficiency of the Pylorus. Incontinentia Pylori.

When the stomach is inflated with air or CO_2, and the gas rapidly distends the entire intestinal tract and is not confined to the stomach, it may be inferred that the pylorus is incompetent. This may be transitory or permanent. Where, through pyloric carcinoma, the musculature has been destroyed, or has lost its contractility, there may be at the pylorus a hard, unyielding ring, allowing free passage into the duodenum. Imperfectly digested food entering the intestines through a constantly patent pyloric orifice may light up intestinal catarrh and diarrhœa.

(*e*) Peristaltic Unrest of the Stomach.

In neurasthenia, anæmia, pyloric obstruction, the peristaltic movement of the stomach becomes exaggerated, visible to the naked eye, and also causes in the patient disagreeable sensations of unrest in the stomach. Nervousness and emotion seem to increase it, as does, at times, the presence of food. The active peristalsis may extend to the intestines. In such a case the patient's abdomen, especially if he be emaciated, looks truly like the waves of the sea, with their ceaseless undulations. This condition is often spoken of as *Kussmaul's peristaltic unrest*, as he first gave an accurate description of it.

SENSORY NEUROSES.

Gastralgia.

Severe paroxysmal pain in the epigastrium may be caused by organic disease, as ulcer; by disease of the central nervous system, as locomotor ataxia (gastric crisis), or may be apparently functional. To this latter form the term gastralgia is more strictly applied.

It is commonly met with in the anæmic or chlorotic or neurasthenic. The pain is sudden in onset, with no dietary error to explain its occurrence, is rarely attended by vomiting. Pressure or the taking of food will at times give relief. In some cases hyperacidity is present, perhaps as a causal factor.

Gastralgia is at times one of the most difficult affections to recognize. Not alone must we aim to discriminate between this pain, functional in character, and that of an organic disease of the stomach, as ulcer or cancer (*vide* page 102), but we must remember that pain originating in organs even at a considerable distance from the stomach may be referred by the patient to the epigastrium. Renal colic and perforative appendicitis, if thought of and examined for, will rarely be overlooked. A commencing pericarditis or diaphragmatic pleurisy, especially if the latter comes on suddenly, as at the commencement of a pneumonia, may cause complaint of severe epigastric pain. Investigation must also be made as to the possibility of lead-poisoning being the cause of the pain. Muscular rheumatism and intercostal neuralgia can be recognized by the superficiality of the pain, the tenderness on light pressure, the existence of like pains elsewhere, and the tender points of intercostal neuralgia.

Gall-stone colic is perhaps oftener than any other disease called gastralgia. The symptoms are often very confusing. Yet when we consider the exact location of the pain and tenderness—to the right of the median line—the vomiting, the history of previous attacks followed by jaundice, we may usually reach the correct conclusion, confirmed oftentimes by subsequent jaundice or the detection of gall-stones in the feces.

SECRETORY NEUROSES.

The three forms of secretory disturbance can be recognized only by chemical examination of the gastric juice. Exact clinical pictures of each type of the disease have not yet been drawn.

(a) Hyperacidity.

This may occur periodically, as in locomotor ataxia, or be chronic. Gnawing pains in the stomach, headache, acid pyrosis, and the vomiting of a clear fluid, strongly acid and irritating to the throat, mark the attacks. Digestion is usually markedly impaired. Quantitative tests show increased acidity of the gastric juice.

(b) Hypacidity—Subacidity.

Aside from the hypacidity of chronic gastritis and carcinoma there are cases where without organic change in the stomach the acidity is below normal or even zero (anacidity).

Anorexia, malaise, gastric uneasiness are the symptoms usually noted. It occurs oftenest in neurasthenics and sufferers from "nervous dyspepsia."

(c) Hypersecretion.

Where an excessive amount of gastric juice is secreted, and this even during periods of fasting, as at night, the term hypersecretion has been applied. In Reichman's case there could be obtained from the stomach in the morning 180 to 300 c.cm. of gastric juice with good digestive power.

MIXED NEUROSES.

Nervous Dyspepsia.

Digestion is usually naturally performed, and yet these patients, markedly neurotic, are sufferers from gastric distress, anorexia, eructation, nausea, vomiting. Some of the worst cases are associated with neurasthenia with its manifold symptoms. The diagnosis is made by the exclusion of organic disease and the evident neurotic temperament of the sufferer.

DISEASES OF THE INTESTINES.

ACUTE CATARRHAL ENTERITIS.

Theoretically it is possible to distinguish an acute catarrhal inflammation of the various anatomical divisions of the intestinal tract, *e. g.*, duodenitis, jejunitis, ileitis, colitis. Practically there is usually more than one division involved, and, even though the inflammation be limited in extent, the difficulties of diagnosis are so great as to render it impossible accurately to define the portion of the bowel affected. When the upper portion of the intestinal tract is involved there is usually also a catarrhal inflammation of the stomach which may be secondary to the enteritis, or *vice versa*.

The same causes that produce gastritis operate to produce intestinal catarrh. In the production of the primary inflammations, mechanical and chemical causes are most widely operative. Poorly masticated food, food that is indigestible, through its inherent nature, or undigested because of perverted gastric function, may, mechanically, produce irritation and inflammation of the mucosa of the bowel. Under the head of chemical causes must be included not only foods, drinks, medicines, poisons that have a direct irritating influence, but also such substances, as, failing of proper stomach digestion, enter the intestine, having undergone organic acid fermentation, charged with noxious gases, chemical compounds, and bacteria. Entrance of bacteria into the intestine, with the resulting irritating chemical products, produces some of the most serious and alarming forms of enteritis, such as cholera morbus and cholera infantum, where there is profound systemic intoxication with bacterial products. Catarrhal inflammation of the intestine is often secondary to disease in other organs, *e. g.*, the stomach, as just shown, the liver or lungs, where disease, as cirrhosis or emphysema, may cause portal obstruction, and thus engorgement of the intestinal vessels. The latter condition, while, perhaps, not the exciting cause, is often a predisposing cause of enteritis, permitting readily, the operation of the causative factors just mentioned. A catarrhal inflammation accompanies organic disease of the wall of the intestine itself, as ulcer, carcinoma. Acute infectious diseases are, at times, accompanied by intestinal catarrhal inflammation. And in uræmia, perhaps from elimination of toxic substances through the intestine and irritation of the bowel, diarrhœa may be extreme.

Sudden changes in temperature, exposure to cold, great fear or anxiety, will bring on diarrhœa, the commonest sign of enteritis. Whether some of these cases, especially the so-called "nervous diarrhœa," can be classed as inflammation, is questionable.

The diagnosis of enteric catarrh rests largely upon the existence of diarrhœa. Increased peristalsis, deficient absorption of fluids, or increased watery secretion into the bowel, may be productive of these frequent, loose stools. All these factors, productive of diarrhæa, may exist in the small intestine, causing the thin fluid contents to be hurried on into the large intestine, and yet diarrhœa not result, provided the colon be not involved, so that the delayed passage of the intestinal contents may here permit of absorption of the greater part of the fluid portion. Acute catarrhal inflammation may, therefore, exist in the small intestine and there be no diarrhœa. Involvement of the large intestine, however (colitis), means frequent, watery discharges. In typhoid fever where there is constipation, it is probable that the large bowel is healthy, while profuse diarrhœa usually means ulceration and inflammation of the cæcum and ascending colon. The number of stools varies from three or four to twenty or thirty, in twenty-four hours. The amount passed at stool depends on the amount of fecal matter in the bowel at the commencement of the attack, the amount of food and drink taken during the illness.

Examination of the stool is of great value in determining the location of the inflammatory affection and its severity. Where the upper small intestine is involved there is found imperfectly digested food, especially starches, and if the stomach digestion is also imperfect, fragments of undigested meat. Diarrhœa, with a large amount of undigested food, is spoken of, at times, as "lienteric diarrhœa." Bile hurried through the small intestine, fails of reabsorption, and often appears in the stools in children, giving the discharges a grass-green color. Mucus is seen, intimately mixed with the particles of food. The microscope may be necessary to show the small masses of mucus which, as well as the cylindrical epithelium and the round cells, may be pigmented (usually yellow) from the bile. If no inflammation of the colon exists the feces may be formed into a mass, and the discharge not be diarrhœal in character.

Where the colon is involved, frequent watery passages are the rule; the mucus is in lumps or stringy masses, visible to the naked eye. Where only the lowest part of the colon is involved there may have been time for the absorption of much of the fluid from the colon, and a fecal cylinder may have been formed, which is coated with mucus as it passes through the inflamed lower portion. Streaks of blood may be seen in the passages of enteritis.

Pain is seldom absent. It is often of sudden onset, griping or colicky in character, causing the patient to double himself up and to make pressure over the abdomen. This fact alone, will often enable us to distinguish the abdominal pain of colic from that of peritonitis, where the patient keeps the abdomen as quiet as possible and avoids all weight or pressure over the inflamed part. After an attack of enteric catarrh there is often for a day or two, after the violent peristalsis has ceased, a soreness over the bowels that may cause the patient some distress and anxiety, lest the illness has not entirely vanished.

Rumbling of the bowels and a moderate degree of tympany are common. Fever may be as high as 102°, or, in children, higher. Gastric disturbances are common, the same cause operating to produce irritation of both gastric and intestinal mucosæ. Headache, backache, pains in the bones and joints, malaise may be marked in various degrees.

(a) Gastro-duodenitis.

History of dietary indiscretion, exposure to cold, etc. Anorexia, nausea, vomiting, epigastric pain, fever as in gastritis; tenderness over the upper zone of abdomen, and not limited solely to gastric region; diarrhœa rare. Diagnosis may be confirmed by the symptoms and signs of catarrhal jaundice in about three days.

(b) Catarrhal Inflammation of the Jejunum and Ileum.

Pain, borborygmi, malaise, etc., as before described. No diarrhœa unless cæcum or colon involved. Stools bile-stained, with undigested food, mucus in small lumps. Many epithelial cells (cylindrical) and round cells. Tenderness over centre of abdomen.

(c) Catarrhal Inflammation of the Large Intestine.

Diarrhœa marked. Mucus in large masses. If no coincident inflammation of small intestine food well digested. Formed fecal masses coated with mucus if only the lower portion of the large intestine is involved. Tenderness over region of the colon, which is often tympanitic. Where the rectum is involved, *proctitis*, severe pain, straining at stool with but a small amount of fecal matter (tenesmus). Digital examination, or specular examination, shows tender and inflamed rectum in the latter condition. Dysentery, with its small stools and marked tenesmus, gives the characteristic symptoms of proctitis.

(d) Cholera Morbus—Cholera Nostras.

Probably microbic in origin. At times a history of some error of diet, at other times no such history. Onset sudden with vomiting and purging, the stools soon becoming thin and watery. There is pain not alone in the stomach and bowels but in the muscles of the arms and legs, which cramp at times, as in arsenic poisoning. The patient becomes weak, even to collapse, the skin is cold, the pulse small, the countenance pinched and dusky, the voice husky. The picture is much like that of Asiatic cholera, and if the latter epidemic be prevailing a bacteriological examination of the dejecta may be necessary to determine the true character of the disease.

(e) Diarrhœa of Children.

Sudden vomiting, pain, fever, restlessness; even convulsions may be caused by child eating some indigestible article of food, as grapes, bananas, etc. The increased peristalsis hurries the contents through the bowel, and there are numerous passages, bile-stained and containing the undigested *materies morbi* as well as curds of milk. With the evacuation of the bowels, especially if aided by some mild cathartic, as castor oil, the trouble usually ends, and the child that was seen in the evening with flushed face, slight delirium, frequent stools, occasional vomiting, temperature 104°, may in the morning be playing about the room as though nothing had happened. In other cases a more lasting subacute catarrh is started in this way.

Cholera morbus in the adult has its counterpart in the **cholera infantum.** Bacterial in origin, it is met with oftenest in the artificially fed and during the hot weather of summer. It seems to be a profound toxæmia. The attack is sudden and severe from the beginning. Vomiting is almost incessant, even the water that is given the thirsty child being ejected. The frequent odorous, feculent stools soon give way to copious, odorless, serous discharges. While the rectal temperature is elevated the skin is cold and clammy. The eyes are sunken, the fontanelles depressed, emaciation is extremely rapid. The baby may cease its moaning and whining and lie for hours in a stupid, semi-comatose state. Death may occur within twenty-four hours, or after several days. The diagnosis is clear, with the history of artificial feeding, the sudden onset with marked collapse, incoercible vomiting and frequent, copious watery discharges. If Asiatic cholera prevails the spirillum of Koch is to be sought for before a definite knowledge of the nature of the case in hand can be obtained.

An **acute entero-colitis**—follicular dysentery—attacks children, especially in summer, at times, with some error in the diet as an apparent exciting cause, and again, with no discoverable etiological factor. Diarrhœa, the stools containing blood and mucus, temperature, moderate vomiting characterize the disease. It lacks the extremely sudden onset, the collapse and incessant vomiting and purging of cholera infantum. After several days, or even weeks, recovery occurs, or a subacute inflammation, with occasional exacerbations, supervenes. Where the rectum is involved, bloody, mucous stools, small in amount, are passed frequently, and with pain and marked tenesmus.

MEMBRANOUS ENTERITIS.

Membranous enteritis is a rare chronic colitis in which, at times, with pain, colonic tenderness, tenesmus, long strings of mucus, or even casts of the bowel are passed.

INTESTINAL ULCERS.

Ulceration of the intestine may accompany typhoid fever, dysentery, tuberculosis, or syphilis. Follicular ulceration is often met with in the acute entero-colitis of children, as are ulcers of the colon in adults with chronic colitis. Constipation may be attended by the stercoral, malignant growth, by a cancerous ulcer. Perforation into the bowel, as from a neighboring abscess, will be accompanied by ulceration.

The diagnosis of some of these conditions is extremely difficult. The most valuable signs and symptoms are diarrhœa, the stools containing pus, shreds of tissue and blood. Severe hemorrhage may occur. Pain may be localized when the ulcers are few. Perforation may occur.

CHRONIC CATARRHAL ENTERITIS.

The continuance in operation or the repeated action ot causes productive of acute intestinal catarrh may produce a chronic catarrh. Following dysentery, malaria, typhoid fever, there is occasionally this condition. Diarrhœa is the most prominent symptom, and the disease is often spoken of as **chronic diarrhœa.** Usually, the large intestine is most markedly involved. If the small intestine be likewise seriously affected, lientery may result. Rarely, chronic ulcers are present. The stools contain mucus, often pus; shreds of tissue, or even blood may be found. Cases are met with in which constipation alternates with diarrhœa. The general health, in some cases, seems but little affected. In others, evidences of malnutrition are marked. Perversion of appetite, coated tongue, borborygmi, occasional colic are trifling, as compared to the nervous derangement. Sufferers from chronic diarrhœa are often irritable, morose, hypochondriacal, even suicidal. The importance of an examination of the stools should be remembered, not alone because of its bearing on diagnosis, but because of the great aid it gives in therapy, showing, usually, the portion of the bowel most affected, the kinds of food most poorly digested, etc. The possibility of a chronic diarrhœa being due to tuberculosis of the intestine must always be remembered.

CONSTIPATION.

The recognition of this condition is seldom difficult. It is to be remembered, however, that constipation, like diarrhœa, its opposite, is, in reality, but the expression or symptom of some diseased condition, either primary or secondary, of the intestine. Usually, there is deficient peristalsis through depressed innervation, depending upon some alteration in the intestine itself, or upon some more widely operating cause, as anæmia, or wasting diseases. Lack of fluid in the bowel, through failure of supply, or through excessive absorption, may cause constipation.

The recognition of the cause is the clew to the treatment.

General symptoms vary. Some patients, if they miss the daily evacuation of the bowels, suffer from headache, depression of spirits, coated tongue, general malaise, dizziness, cold feet. This is probably a mild toxæmia (copræmia). It must not be forgotten, that, while a single movement of the bowels daily is the rule for the majority of people, there are those who, with all the evidences of health, go two or three days without defecation, while others will have two or more normal movements every day.

The cases most difficult of recognition are those in which the patient, with ill-defined symptoms of disease, declares that there is no constipation, the bowels moving with clock-like regularity every day. Yet there may be retention of fecal masses in the folds and pouches of the bowel, only demonstrable by careful palpation of the abdomen, or by flushing the colon daily with large amounts of warm water. This latter procedure will finally succeed in freeing these masses which appear as hard, black lumps in the stool.

APPENDICITIS.

Inflammation of the vermiform appendix may be **catarrhal**, accompanied by **adhesive peritonitis**, by **localized suppurative peritonitis** or **abscess**, or by **diffuse suppurative peritonitis**. Clinically, such distinctions cannot be made, and we can only fairly well distinguish a catarrhal form, and the perforative form with (*a*) local or (*b*) general peritonitis.

It occurs oftenest in boys and young male adults. Fecal concretions are often present as an exciting cause. Numerous cases of catarrhal appendicitis and of appendicitis with local, plastic, or even suppurative peritonitis, recover unrecognized by patient or physician, because of the triviality or total absence of symptoms. Autopsy may furnish the only proof of a previous appendicitis.

Catarrhal appendicitis seems, at times, to have the paroxysm excited by a blow, a strain, overindulgence at table. Again, without any assignable cause, there is complaint of pain in the right iliac region, exaggerated on movement of the right leg. There may be some vomiting. Constipation and slight rise in temperature are the rule.

The pain may continue for only a day or two or for several weeks, often recurring after apparent recovery.

Where there is **perforation** the pain is sudden, referred to the right inguinal region, or, at times, to the umbilical; vomiting is common, as is constipation. The countenance is pale and expressive of suffering. The right thigh is flexed and any movement of the psoas muscle causes pain. The temperature may be as high as 103°, or but slightly elevated. Examination reveals tenderness in the right iliac region, by pressure with one finger, found to be often most intense half way between the anterior superior spine of the ilium and the umbilicus—McBurney's point. Tympany may be marked. Greater resistance meets the examining fingers, the muscles often being much more tense on the right than on the left side. Œdema may be noted, and in cases, a swelling made out. The larger the collection of pus the more readily, of course, is a swelling detected. Percussion is of little aid, the colon usually giving its tympanitic resonance, as it overlies the abscess. Rectal and vaginal examination may reveal induration or fluctuation, and in the female, serve to exclude salpingitis.

General peritonitis may result from the perforation of an appendicular ulcer directly into the abdominal cavity, before an adhesive peritonitis has walled it off, or it may result from the breaking down of the adhesions, a local abscess, by the escape of pus into the general cavity, causing a diffuse suppurative peritonitis. The greater area of pain and tenderness, greater tympany, more pronounced shock, and the progressively downward course of the case furnish the evidences of this almost uniformly fatal sequel of appendicitis.

It is to be remembered that perforation of the vermiform appendix without stormy symptoms may occur, and the pus gradually burrow to a region remote from its starting point. The physician may find in these cases an abscess in the region of the kidney, even perforating the diaphragm and causing suppurative pleuritis. Rupture into the bladder, uterus, bowel, portal vein has occurred more than once. General sepsis or pyæmia may start from appendicitis.

It is the consensus of opinion that the use of the aspirating needle for diagnostic purposes is not without serious danger.

A tender, doughy swelling in the cæcal region, with slight fever, pain, constipation, flexion of right thigh, give evidences of **stercoral cæcitis.** The exact pathological condition in these cases, many of which may be due to disease of the appendix, is not clearly understood.

Appendicitis has frequently been called **typhoid fever,** and especially where the onset has not been as sudden as in typical cases. A case carefully watched for a few days will usually clear up any doubts in the mind of the physician. So many of these cases of appendicitis, however, are sent to the hospital with a physician's diagnosis of typhoid, that a tabular differential diagnosis may be in place. It is, perhaps, needless to say that the confusing points are the daily fever, headache, anorexia, coated tongue, pain and tenderness in the right iliac region, tympany and derangement of the bowels, which may be shown by constipation. These symptoms in a young adult and during an epidemic of typhoid, quite easily mislead one into a mistake.

Differential Diagnosis.

Typhoid Fever.	Appendicitis.
1. Onset gradual; anorexia and headache complained of before abdominal pain; latter may be almost absent; vomiting not common.	1. Onset generally sudden, anorexia, headache, etc., succeed the abdominal pain, which is always present to some extent. Vomiting at begining of attack.
2. Pain often elicited only on pressure.	2. Pain more localized than in typhoid.
3. Examination of iliac region shows pain on pressure, usually with gurgling.	3. Examination of iliac region, where pain is usually complained of may reveal swelling, induration, tense muscles, œdema. McBurney's point often found.
4. No flexion of thigh.	4. Involuntary fixation of psoas muscle, flexing thigh.
5. Bronchitis early. Later, splenic tumor, rose-spots, mental hebetude.	5. No bronchitis, splenic tumor or rose-spots. Patient usually bright.
6. Characteristic step-like rise of temperature.	6. Temperature (unless peritonitis spreads) tends to become lower after first few days. Is apt to be more irregular.

Chronic tubercular appendicitis and cæcitis, with exacerbations of acute inflammation, may give the symptoms and signs of a recurrent catarrhal or ulcerative appendicitis with perforation.

The diagnosis of appendicitis is often aided by a history of previous similar attacks. Recurring forms are met with in which patients have suffered several times from pain in the right iliac region, vomiting, fever, tenderness, induration, etc., and after a few days in bed have recovered. These attacks, often regarded as "inflammation of the bowel," point in a woman in the direction of a salpingitis, in man excite suspicion of appendicitis.

In conclusion, I may state Osler's words: "Almost without exception, sudden pain in the right iliac fossa, with fever, localized tenderness, with or without tumor, means appendix disease."

INTESTINAL OBSTRUCTION.

Acute intestinal obstruction, though properly, in the majority of cases, an affection to be dealt with by the surgeon, comes so often first under the eye of the physician that it is upon him that the grave responsibility of diagnosis rests; grave responsibility, because if untreated, most of these patients die a painful death, and grave, too, because a tardy diagnosis makes even the faultless operation of the surgeon fruitless. Gangrene, peritonitis, shock, may have come on while the physician hesitates as to what is the matter. As Hunter McGuire says, "Many of the causes of death following laparotomy (for obstruction) should be ascribed not to the fact that the knife was used but to the fact that it was used too late."[1] The limits of the present volume permit only an outline of the most important features in diagnosis. The articles of Leichtenstern, Treves, Senn, Fitz can be consulted for elaborate and minute details.

Intestinal obstruction, signifying the impassableness of some portion of the intestinal tract, may be due to causes that may be classified as follows:

1. Abnormalities in the contents of the intestine.
2. Alterations in the position of the intestines.

(*A*) Hernia (strangulated).

a. External, *e, g.*, inguinal, femoral, umbilical.

b. Internal, where gut passes through.

(1) An opening or pocket in the peritoneum normally pres-

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[1] Pepper's System of Medicine, vol. ii, p. 866.

ent—*e. g.*, the foramen of Winslow; duodeno-jejunal or subcæcal fossæ, diaphragmatic defects.

(2) Abnormal slits or openings in the mesentery or omentum.

(3) Abnormal bands or pseudo-ligaments, the result of a previous peritonitis, or the remains of embryonic structures. (Meckel's diverticulum).[1]

(*B*) Volvulus (twists) and knots.

(*C*) Intussusception.

3. Pathological changes in the wall of the intestines—*e. g.*, carcinoma, polypus, cicatricial tissue from preceding ulcer, congenital atresia.

4. Compression from without, as by neoplasm, aneurism, abscess.

5. "Ileus paralyticus."

1. Fecal impaction (coprostasis) is the commonest of these causes. Feces may accumulate in any portion of the bowel, though oftenest in the cæcum or rectum. Gall-stones, bunches of intestinal worms, peach-stones, enteroliths, may form the obstructing body or its nucleus.

2. This group includes the larger number of cases. Internal herniæ and strangulation are pathologically identical with external. The bowel may be caught in any of the normally present pouches and openings of the abdominal cavity, or, as is often the case, bands of adhesions, the result of previous peritonitis, plastic, tubercular, or after an operation, may form openings and pockets in which the intestine becomes incarcerated, with resulting stasis, œdema, gangrene, perforation, peritonitis.

Twists and knots closing the lumen of the bowel are favored by a long mesentery and increased peristalsis, and occur oftenest in the region of the sigmoid flexure.

Intussusception, or invagination, is the slipping of the upper portion of the bowel (oftenest the ileum) into the lower (the cæcum and colon). Rarely the lower slips into the upper. Figures 20 and 21 illustrate the position of the gut in this condition. Blood stasis due to constriction at the neck may be followed by œdema and necrosis of the entering part, resulting in spontaneous recovery if adhesions have formed sufficient to shut off the abdominal cavity. More often the untreated case leads to gangrene and peritonitis, usually from perforation.

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[1] The remains of the embryonic omphalo-mesenteric duct through which the intestine communicated with the yolk-sac. If the end be attached to the abdominal wall or mesentery, instead of being free, a ring is formed through which a loop of bowel may pass. The diverticulum usually springs from the ileum near the cæcal junction.

3. Malignant or benignant growth, cicatricial tissue from preceding inflammatory changes involving the submucosa, may lead to chronic obstruction. Acute obstruction may take place by the lodgment at the point of narrowing of fecal matter, gall-stones, etc., or, by the violence of the peristalsis excited in the efforts of the intestine to force its contents through the constriction, a volvulus is produced.

FIG. 20.

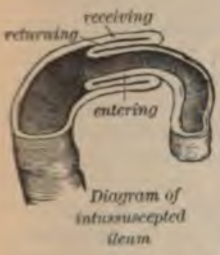
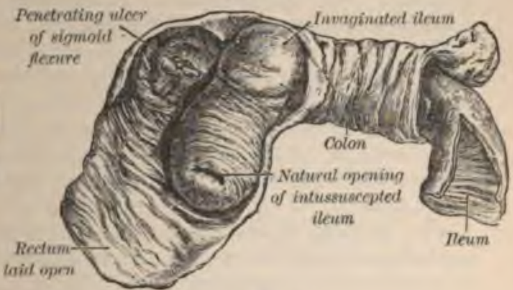


FIG. 21.


Penetrating ulcer of sigmoid flexure
Invaginated ileum
Colon
Natural opening of intussuscepted ileum
Ileum
Rectum laid open

Intestinal intussusception. (McGUIRE.)

4. External compression is a rare cause of acute obstruction.

5. "Ileus paralyticus," the dynamic obstruction of Leichtenstern is, in contradistinction to the other mechanical forms, a functional obstruction. Paralysis of the musculature may follow the reduction of a hernia or volvulus; it often accompanies a local peritonitis, as from appendicitis or salpingitis, or a diffuse form of peritoneal inflammation giving rise to many of the clinical features of mechanical obstruction—*e. g.*, constipation, tympany.

The classical symptoms of acute obstruction are abdominal pain, constipation, vomiting, tympany, shock. In this disease, as in so many others, one or more of these symptoms may be lacking or modified, and still the diagnosis be possible.

Pain often comes on suddenly. It is abdominal, and at first colicky. It increases in severity, and if peritonitis occurs becomes more diffuse and lancinating.

Vomiting is commonly present early. Bile and mucus are ejected, and after some hours or days fecal matter. Stercoraceous vomiting is one of the most reliable evidences of intestinal obstruction.

Constipation may be present from the beginning of the attack, though the contents of the bowel below the point of obstruction may be passed. In intussusception, bloody passages and tenesmus are present in most cases. In other cases of obstruction there is usually complete constipation, neither feces nor gases escaping.

Unless the obstruction is high up, **tympanites** is marked, often to an extreme degree. In duodenal obstruction, on the contrary, the flattened coils of the lower intestine may allow a sinking in of the abdomen. Exaggerated peristalsis is frequently seen through the abdominal wall.

The amount of **shock** varies. It may be extreme from the start. Auto-intoxication probably accounts for some of it. Rapid, feeble pulse, clammy skin, anxious, livid countenance, hiccough, subnormal temperature, give evidence of the serious character of the trouble. Peritonitis occurring in connection with the obstruction adds to the intensity of these symptoms.

The **location of the obstruction** can in many cases be definitely ascertained, while in others it cannot be fixed other than by exploratory laparotomy. The site of the pain, the point of greatest tenderness should be sought for. Palpation may reveal a swelling, as in intussusception or fecal impaction. Excessive tympany speaks for obstruction low down in the intestine, especially the colon, failure of tympany for obstruction high up. With the centre of the abdomen tympanitic and the sides not, the ileum is usually the seat of the difficulty. Examinations *per rectum et per vaginam* will furnish aid in those cases where the cause is intra-pelvic. In obstruction of the ileum indicanuria is marked. Obstruction high up in the duodenum or obstruction in the colon fails to cause an increase in indican in the urine. Too much reliance, however, cannot be placed on the finding of indican in the urine, for in some cases where the large intestine is involved, especially where there is coincident peritonitis, the urine is rich in indican. The failure to find indican is of more diagnostic value, pointing to the large intestine as the seat of trouble.[1]

[1] Stenosis of the small intestine permits the decomposition of certain albuminous substances and allows putrefactive changes to take place, the result of all which is the formation of chemical compounds absorbed and excreted by the urine as indican. If the obstruction be in the large intestine, however, the changes in these albuminous substances do not take place as the albuminoids are properly digested in the small intestine.

The injection of air, or, better still, hydrogen gas, into the intestine *per rectum*, may furnish a valuable and sure means of determining, not alone the question as to the patency of the intestine, but as well the location of the obstruction. Hydrogen gas injected continuously and slowly from a large gas-bag through a tube passed to the sigmoid flexure, and under a pressure of two pounds to the square inch, will pass the ileo-cæcal valve. Used as an early means of diagnosis before necroses or inflammation have weakened the intestinal wall, this method is safe.[1]

In some cases laparotomy is justifiable as a means of diagnosis as well as of cure.

It is usually true that the higher up the stenosis the more pronounced the evidences of shock.

The character of the obstruction is important as it influences largely the remedial measures employed and the prognosis. Unfortunately it is too often impossible accurately to decide as to the nature of the operating cause.

Impacted feces may give a history of chronic constipation. Pain, vomiting, shock, are usually only moderately well marked at the beginning and only gradually grow worse. Palpation of the abdomen or rectal examination will generally reveal the presence in the colon or rectum of the offending mass. The possibility of gall-stones being the cause of the difficulty is to be remembered and inquiry instituted as to attacks of hepatic colic, jaundice, etc.

Intussusception occurs oftenest in children, comes on suddenly, with severe pain and marked shock. Tenesmus and bloody stools, even diarrhœal in character, are more common than constipation. A swelling, "sausage-shaped," tender to pressure, can be felt usually in the region of the cæcum and colon. The invaginated bowel at times can be felt by rectal examination, or even seen as it protrudes.

When there is strangulation by **bands**, or by the intestine being caught in some pocket or ring, the history may show a previous peritonitis or operation. The patients are as a class adults. Pain is sudden and severe, vomiting and prostration marked, tympany sometimes slow in developing.

Volvulus occurring in adults comes on with sudden severe pain. Usually found at the sigmoid flexure, where there is tenderness, it causes vomiting and prostration less early than is met with in obstruction higher up. If unrelieved fecal vomiting occurs, and shock is marked. Constipation is absolute in volvulus.

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[1] Senn: Intestinal Surgery, pp. 12, 88, et al.

The following table of differential diagnosis of the commoner forms of acute and chronic obstruction is made by Shattuck, and based upon the essay of Treves:

ACUTE INTESTINAL OBSTRUCTION.

Table of differential diagnosis.

	Strangulation by bands or through apertures (25 per cent. of all cases of acute obstruction).	Volvulus of colon.	Acute intussusception.
Age, sex,	Young adults; rare after forty.	Males as 4 : 1; 40 to 60.	More than 50 per cent. under 10 years.
History,	Previous peritonitis in 68 per cent.; previous attacks of obstruction in 12 per cent.	Previous constipation.	Usually negative.
Onset,	Sudden in 70 per cent.	Sudden.	Sudden in 75 per cent.
Pain,	Early, severe, continuous, with exacerbations.	Early, less severe, intermittent, becoming constant with exacerbations.	Early and severe; increasing and later subsiding; at first paroxysmal.
Local tenderness,	Absent at first, appears later.	Early over-distended coil, and constant.	Common about a tumor.
Vomiting	Early, marked; in 60 per cent. becomes feculent; affords no relief.	Less early, severe, and constant; often affords relief.	Still less early and severe; in 25 per cent. becomes feculent.
Constipation,	Continuous and absolute; no blood.	Early and absolute; no blood.	Absolute; rare: diarrhœa not uncommon; blood in 80 per cent.
Prostration,	Marked.	Rather less marked; may be dyspnœa.	Marked.
Tenesmus,	Absent.	In 15 per cent.	In 55 per cent. and often early.
Abdominal wall,	Flaccid unless peritonitis.	Rigid from early peritonitis.	Flaccid unless peritonitis.
Tumor,	Very rare.	Absent.	In 50 per cent., invagination sometimes felt in rectum.
Meteorism,	Slight, appears about third day.	Early, rapid, increases, is extreme.	Rare unless marked constipation.

NOTE.—No trustworthy conclusions can be drawn from the seat of the pain as to the seat of the obstruction, unless local peritonitis comes on. The pain is usually referred in all

forms to the region of the navel. In complete obstruction the pain is constant, though with exacerbations; intermittent pain shows that the obstruction is partial. Coils of intestine are not visible through the abdominal wall in acute cases.

CHRONIC INTESTINAL OBSTRUCTION.

Table of differential diagnosis.

	Stricture of the small gut.	Stricture of the large gut.	Fecal accumulation.
Age, sex,	Adults.	Adults.	Adults; more common in females; the hysterical, lunatics, hypochondriacs.
History,	Cancer, trauma, tuberculosis; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Cancer, trauma, tuberculosis, dysentery: disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Previous constipation.
Onset,	Gradual.	Gradual.	Gradual.
Pain,	Intermittent.	Intermittent.	Less prominent.
Vomiting	Late, scanty, feculent only toward end of acute attack; may be provoked by food.	Less prominent, rarely feculent or provoked by food.	Late, scanty, rarely feculent, often absent
Constipation,	May alternate with diarrhœa; blood points to cancer.	Form of feces may be altered; blood points to cancer.	Gradually increasing; may be spurious diarrhœa; no blood.
Tenesmus,	Absent.	Often present.	Absent.
Meteorism,	Not marked, unless acute attack.	Often marked.	Late; generally increases with obstruction.
Tumor,	Only in cancer, and then in 30 per cent.	Only in cancer, and then in 40 per cent., may be felt in rectum.	Common and distinctive; most easily felt in cæcum; little or no tenderness sometimes movable, and can be changed in shape.
Coils of intestines	Marked in proportion to emaciation.	Marked in proportion to emaciation.	Rarely seen.

N. B.—In any form of chronic obstruction the symptoms of acute occlusion may suddenly supervene.

DIFFERENTIAL DIAGNOSIS.

From **Acute General Peritonitis.** The diffuseness of the pain, the exquisite tenderness, the early tympany and shock, together with the wiry pulse, hiccough, fever and "facies abdominalis," (pallid countenance, livid ears, sunken eyes and cheeks, pointed nose, anxious expression,) usually render a diagnosis clear. A cause for the peritonitis can often be found in a perforating gastric ulcer, a suppurating Fallopian tube, or an appendicitis. The latter condition is easily mistaken for obstruction, unless a careful subjective and objective examination be made. Much help is often derived in the attempt (sometimes futile) to differentiate a general peritonitis from an obstruction by remembering that in peritonitis peristalis is stopped, in obstruction usually exaggerated, the working of the intestinal coils often being seen and felt through the abdominal walls. The early occurrence of diffuse pain, with fever (though this may be lacking), in peritonitis is not to be forgotten. It is also to be remembered that peritonitis may supervene in a case of obstruction.

From **Intestinal Colic, Lead Colic with Constipation.** Severe abdominal pain, constipation, vomiting, especially in a painter, may simulate obstruction from mechanical causes. A knowledge of the occupation, the blue line about the gums, wrist-drop, history of previous attacks will put us on our guard. It is more perplexing to have a genuine case of obstruction (as from volvulus) in a painter, with the blue line and the other evidences of lead poisoning present. Unless most carefully examined and watched, the serious error may be committed of letting the condition pass as lead colic and constipation.

Other conditions that may simulate obstruction are acute enteritis, especially in children, hepatic colic, renal colic, the "incarceration" of a floating kidney, acute hemorrhagic pancreatitis.

INTESTINAL PARASITES.

General Symptoms. In many cases of "worms" symptoms are absent, and the discovery of the parasite in the stools is the first intimation of its existence. In other cases there are fairly well-marked symptoms. Locally there may be a sense of uneasiness, or even pain, in the bowels. Itching about the anus is very noticeable in many cases of thread-worms. Dyspeptic symptoms may be marked, the appetite being capricious, sometimes nausea and irregularity of the bowels being present. Nervous phenomena, such as itching of the nose, starting in the sleep, "night terrors," gritting the teeth, vertigo, palpitation, even convulsions and choreic movements are sometimes seen.

Where intestinal parasites are suspected a brisk purge is in order. The stools following will almost surely contain segments of the tapeworm, if that is the offender, or colonies of pin-worms if they are the intestinal inhabitants.

The commoner varieties of intestinal parasites are the following; more complete descriptions can be found in larger works:

Tape-worms (Cestodes).

Tænia Solium: Pork tape-worm. Head, size of pinhead; has four black cup-like suckers, surrounded by double row of hooklets. Neck, thread-like. Body, five to ten feet long, made up of a colony of white, flat, pumpkin-seed-like segments.

Tænia Saginata. Tænia Mediocanellata: Beef tape-worm. Head, larger than of tænia solium; no hooklets, four suckers, segments fatter; length of worm, five to twenty feet.

Bothriocephalus Latus: European.

Nematodes.

Ascaris Lumbricoides: Round-worm. Pale-pink color, two to twelve inches long, looks like ordinary angleworm. Usually one to twelve in bowel. May enter stomach and be vomited, or may crawl up to mouth, larynx, nares.

Oxyuris Vermicularis: Pin-worm, seat-worm, thread-worm. Looks like bits of white thread, one eighth to one-half inch long. Usually many. Cause itching, may enter vagina and cause irritation there.

Anchylostomum Duodenale: European. Cause of severe and fatal anæmia among miners, brickmakers, tunnel-workers (St. Gothard).

Filaria Sanguinis Hominis: Small, thread-like. Gets into lymph and bloodvessels, where can be seen under microscope, best at night. Symptoms of chyluria, chylous ascites, hæmaturia, lymph-scrotum.

Trichina Spiralis. From pork. Worm works its way to muscles of hog and becomes encapsulated. Uncooked pork swallowed by man, capsule dissolved, embryos liberated, procreation, multitudes of embryos born into stomach and intestine. This event, attended by gastro-intestinal disturbance in man, the host. Pain, nausea, vomiting, diarrhœa, fever.

Embryo by the end of fourteenth day has entered muscles, excited myositis and has coiled itself up. As muscles are invaded they become swollen, hard, painful, very tender. Eyelids swell, face is puffy. Diaphragm and eye muscles often involved, rendering breathing difficult and eye movements painful. Patient has fever, is weak, may reach typhoid state, and die.

Diagnosis: 1. History of eating uncooked pork. 2. Gastro-intestinal disturbance. 3. A few days after the subsidence of symptoms of indigestion, muscular soreness, swelling, etc. 4. Œdema of face. 5. Typhoid state. 6. Excision of a piece of muscle and detection of parasite.

May be confused with typhoid fever or with polymyositis. Distinguished from typhoid by history, muscular swellings and tenderness, swelling of face, absence of eruption, detection of parasite. From polymyositis can, in some cases, only be distinguished by excision of muscle. In these cases there is no history of eating raw pork, no primary gastro-intestinal disturbance, no œdema of face, the parasite is not found in the muscle excised, though myositic changes are marked.

DISEASES OF THE LIVER AND THE BILE-DUCTS.

METHODS OF EXAMINATION.

More aid in the diagnosis of diseases of the liver is furnished by **palpation** than by any other method of physical examination. The palpating hand should be warm and applied with the palm to the abdominal wall. Pressure, gently and firmly made, with the palm flat, will reveal more than a sudden, sharp stroke with the fingers alone. Where ascitic fluid or gas covers the liver, the latter method—"dipping"—will, sometimes, enable the organ to be reached easier than the slower and, ordinarily, preferable one. The abdominal muscles should be relaxed, as far as possible, by the patient's voluntary efforts, by the continuance of natural breathing and by flexion of the knees. One can direct the patient to take a very deep breath and then make forcible expiration. This may succeed in relaxing the abdominal muscles where other measures fail. At times a satisfactory palpation can only be made under an anæsthetic. By palpation we aim to determine, not alone the lower limits of the liver, but the condition of its edge, whether sharp, rounded, nodular; and its surface, whether smooth, or rough and irregular. Points of tenderness, of fluctuation, or of œdema, are also sought for, and the question of the mobility of the organ with the depression of the diaphragm, is to be determined.

Inspection, as an aid in determining the size and shape of the liver, is, in many cases, of negative value. Where tumors or abscesses are large the prominence may be very perceptible. And, even where there is but a moderate degree of uniform enlargement, the transverse ridge across the abdomen, marking the sharp edge of the liver, may be seen, in case the abdominal parietes are thin.

Auscultation furnishes no positive aid, save in cases of localized peritonitis (perihepatitis), usually chronic, where a friction rub is occasionally heard.

Percussion over the liver, covered only by the thoracic or abdominal wall gives flatness. Where the lung shelves over it there is relative dulness, as also below, wherever the liver drops back from the abdominal wall and a coil of intestine works into the gap. In this way, even resonance may be found over the lower ribs, where, usually, there is dulness. Gas loose in the abdominal cavity, may obscure liver dulness, by resonance. The normal liver, in the right nipple line, reaches from the lower border of the fourth rib to the costal arch. From its upper limit to the lower border of the sixth rib, it is covered by lung, and gives on percussion, **relative dulness.** From the sixth rib to the costal arch, there is absolute dulness, or flatness. In the epigastric region dulness extends from one to two inches below the xiphoid appendix. Practically, the costal arch may be looked upon as the limits of the edge of the liver, though there are, in the healthy, variations from this standard, and the erect posture, or a deep inspiration, will depress the liver often as much as an inch. The upper percussion limits of the liver are in the mid-line the base of the ensiform appendix, mammillary line the sixth rib, axillary line the eighth rib, and posteriorly the tenth. Deep inspiration increases the area of relative dulness.

The gall-bladder lies just within the mammillary line, on a level with the junction of that line with the costal arch—at about the ninth costal cartilage.

Confused conceptions as to the mechanism of portal obstruction and of jaundice often lead students into errors of diagnosis, into thinking that jaundice means always a disease of the liver, or, conversely, that every disease of the liver is, of necessity, attended by jaundice. They are puzzled, too, to understand how, in some cases, symptoms of gastro-enteric catarrh and abdominal ascites are the prominent features of a case that is, they are told, primarily an affection of the liver, *e. g.*, cirrhosis. It is well to call to mind the fact that jaundice is due to the production of more bile by the liver cells than can escape through the biliary passages, and its consequent absorption into the circulation by the hepatic lymphatics and bloodvessels. The most marked degrees of jaundice are, therefore met with where the *liver is healthy* and secreting its normal amount of bile, but, where, at the same time obstruction to the outflow exists in the larger bile passages as from a gallstone, inflammation and consequent swelling of the common duct, or pressure upon this duct by a tumor. In some of the most grave affections of the liver, as cancer, cirrhosis, abscess, jaundice may be slight or absent, as the amount of secreting structure destroyed so lessens the amount of bile produced as to leave the biliary passages, also, perhaps, lessened in number or capacity, still competent to perform their function. A small amount of bile absorbed into the blood may be eliminated by the urine, with no staining of tissues visible to the naked eye. And it is not to be forgotten that jaundice may be hæmatogenous, as in pyæmia. It is to be remembered, then, that jaundice may be present and the liver itself be perfectly normal, and that on the other hand serious disease of this organ may exist without perceptible discoloration of tissues.

Obstruction to the flow of blood through the liver, as in cirrhosis, or out of the liver, as in uncompensated valvular disease of the heart, has practially the same effect that would follow the application of a ligature to the portal vein, with the partial or complete occlusion of the lumen. There is a backing up of blood in the portal system, whose radicles become so engorged as to permit the transudation of serum, or even to rupture. When we remember that these radicles are met with in the peritoneum, in the walls of the stomach, intestines, spleen, we understand how their chronic engorgement causes ascites, gastric catarrh, diarrhœa, escape of blood by vomiting or at stool. The prominent symptoms therefore, in some hepatic disorders, *e. g.*, cirrhosis, passive hyperæmia, may be referred by the patient to the stomach or bowels, and mislead the student, unless he clearly understands how these symptoms may be but evidence of a primary disease in the liver.

CIRCULATORY DISTURBANCES OF THE LIVER.

1. Anæmia.

Anæmia of the liver can only be inferred from the existence of general anæmia.

2. Active Hyperæmia.

This cannot ordinarily be diagnosed. It occurs during acute infectious diseases and, physiologically, after meals. In India, climatic influences seem to bring about an hepatic hyperæmia. It is an increase in the amount of blood supplied to the organ.

3. Passive Congestion.

Passive congestion is due to an obstruction to the outflow of blood through the hepatic veins, which are valveless, close to the heart, and whose blood is under a low pressure.

The causes may be grouped as:

1. In the *veins* themselves (rare) as narrowing, due to periphlebitis (hepatic), perihepatitis.
2. *Pressure* on the veins as from aneurism, new growth (in retro-peritoneal glands).
3. Disease of the *heart*, especially right heart dilatation with venous stasis, as from mitral or tricuspid disease or congenital defects (morbus ceruleus).
4. Disease of the *lungs* as emphysema, fibroid phthisis, chronic bronchitis.

Symptomatically, there are first, the symptoms of the primary disease, often the most prominent; secondly, the sense of weight and dragging in the right side, from the heavy liver, often pain, perhaps from a distended capsule or ligaments put upon the stretch, frequently the right decubitus; thirdly, the evidences of portal obstruction, as anorexia, nausea, diarrhœa, hæmatemesis, melæna, ascites, hæmorrhoids. Pressure of the engorged sub- and intra-lobular veins may obstruct enough bile capillaries to cause a moderate jaundice. The right hypochondrium is prominent, the liver is found uniformly enlarged, reaching, perhaps, even to the umbilicus, its edge is sharp, or slightly rounded, but smooth, its surface smooth, often tender on pressure. Uniform expansion of the liver with each heart-beat—liver pulse—is sometimes seen and felt. There is no enlargement of the liver, in which such sudden variations in size from day to day occur. A profuse hemorrhage, a few days' rest in bed, improved heart action, as through digitalis, may cause a perceptible lessening in the size of the organ, while sudden enlargement may follow exacerbations of heart difficulty.

The **diagnosis** then is made upon (1) a knowledge of the cause, (2) the symptoms and signs of enlargement and portal obstruction, (3) variations in size from day to day.

JAUNDICE—ICTERUS.

Hæmatogenous jaundice is met with in conditions such as pyæmia, where there is an excessive destruction of red-blood corpuscles. Other forms are **hepatogenous**, and are due to obstruction to the free outflow of bile. This may be in the smaller ducts—intra-hepatic—as occurs at times in passive congestion, or more often, extra-hepatic, in the larger biliary passages. The obstruction may be (*a*) within the duct—a foreign body, as gall-stone, ascarides, echinococcus, inspissated mucus; (*b*) in the wall of the duct, as scars, new growth, inflammatory swelling; (*c*) outside the duct by pressure, as new growth, *e. g.*, in the head of the pancreas, aneurism, wandering kidney. Mental depression has been said to cause jaundice, just how is not well understood.

The symptoms, naturally, fall under three heads, those due to (1) the original disease or causative factor, (2) the absorption of bile, (3) the absence of bile from the intestine.

1. Here there is, of course, the greatest variation, according as the exciting cause of the obstruction is an impacted gall-stone, carcinoma of the pancreas, gastro-duodenal catarrh, etc. This latter form is often spoken of as **catarrhal jaundice.** Errors of diet, cold, malaria give rise to catarrhal inflammation of the stomach and duodenum. The inflammation may extend up the common duct. As a result, there is swelling of the duodenal mucosa and that of the duct, inspissated mucus forms a plug near the orifice of the duct, and the bile, under its low pressure, is unable to escape. Jaundice appears about four days following the obstruction, and often after the anorexia, nausea, epigastric pain and tenderness, and slight fever have disappeared.

2. Biliary absorption causes the (*a*) tingeing of the conjunctivæ first, later the skin. The color varies through all the shades of yellow. (*b*) The urine contains bile pigments.

The sweat also contains bile. (*c*) The impress of the cholæmia upon the nervous system is shown by an abnormally slow pulse, by irritability of temper, despondency, drowsiness, or, at times, insomnia. In severer cases and those of long standing, a typhoid state is reached, and the patient, feverish, with rapid pulse, becomes delirious, often passing into a fatal coma, which may be preceded by convulsions. It is to this latter group of phenomena that the term cholæmia has been applied. (*d*) There is often an intense itching of the skin, and, in some cases, urticarial wheals or furuncles are seen. Sweating is common, the sweat staining the linen yellow.

3. The absence from the intestine of bile, which acts as a laxative and antiseptic, causes constipation, fetid, pasty, grayish stools. Rarely, there is diarrhœa.

Hæmatogenous jaundice is recognized by the absence of any cause of obstruction, by the presence of bile in the intestines, the stools lacking the pasty consistency, fetid odor and gray color of obstructive jaundice, by the slight degree of yellowness of skin and conjunctiva, and by the evidences of septicæmia or toxæmia. It is found in acute yellow atrophy of the liver, in yellow fever, malaria, pyæmia, poisoning by various chemicals, as phosphorus, mercury, chloroform. The so-called cholæmic symptoms are more liable to occur in this form of jaundice.

GALL-STONES. CHOLELITHIASIS.

Gall-stones may be present in the intra-hepatic bile-ducts, in the cystic duct, or in the gall-bladder, and cause no symptoms calling attention to their existence; or somewhat vague and indefinite complaints as to pain and uneasiness in the right hypochondrium may be made, but nothing characteristic of their presence be manifested. At times the occurrence of complications, as perforative peritonitis, empyema of the gall-bladder, abscess of the liver, is the first evidence of cholelithiasis.

The majority of patients with gall-stones, however, suffer at some time or other from attacks of pain known as gall-stone or hepatic colic, caused, at least in the greater number of cases, by calculi working their way through one of the larger ducts, notably the common duct. The pain is usually sudden in onset, severe, and attended by more or less shock shown by the rapid pulse, pale countenance, cold perspiration, and feeling of great weakness. The pain is referred to a point a little to the right of the median line, and just below the costal arch. Here is the greatest sensitiveness on pressure, and from this point the pain often radiates toward the epigastric and umbilical regions, and toward the right scapula and right shoulder. Vomiting is a common occurrence, and there may be an initial rigor. The pain may last for only a few minutes or for many hours, gradually growing worse, only ceasing when the gall-stone passes through the canal, dropping into the duodenum, or when it falls back, perhaps into the gall-bladder or some diverticulum of the duct. Frequently heroic doses of morphia or the inhalation of chloroform are necessary to render the pain endurable. Following the passage of a gall stone there is often, through the consequent inflammatory swelling of the walls of the common duct, an obstructive jaundice. This is of the greatest diagnostic importance. Examination of the stools for calculi should always be made where gall-stones are suspected, not alone to prove their presence, but also to form some estimate as to their number and character. Multiple calculi have, by mutual attrition, smooth, plane surfaces or facets, and distinct angles. A solitary calculus is rounded or oval and not angular. To examine for calculi the feces should be mixed with water and then filtered through a sieve.

Tenderness over the region of the gall-bladder can often be made out even in the intervals between the attacks of colic. The gall-bladder distended with calculi can at times be felt, and even the friction rub of one stone upon another has been felt and heard. Gall-stones are found oftenest in women and those of sedentary habits. The calculi may vary in number from one to several hundred, and in size from that of a millet seed to a hen's egg.

Impaction of a gall-stone in the cystic duct is followed by **dropsy of the gall-bladder.** Jaundice is not present. An elastic, rounded swelling, at times the size of a fœtal head, is made out in the region of the gall-bladder. This, with the history of colic, the lack of cachexia and fever would suggest hydrops of the gall-bladder.

Impaction of a calculus in the hepatic or common duct causes chronic jaundice often of extreme degree, and at times attended by pronounced or even fatal cholæmia. Catarrhal or suppurative cholangitis may follow. In some cases there is a peculiar complexus of symptoms attending gall-stones and catarrhal cholangitis. Osler makes the following synopsis of these symptoms: (*a*) "Ague-like paroxysms, chills, fever and sweating; (*b*) jaundice of varying intensity, which persists for months, or even years, and deepens after each paroxysm; (*c*) at the time of the paroxysms, pains in the region of the liver, with gastric disturbance." This is the **intermittent hepatic fever** of the French.

Hepatic colic may be mistaken for *gastralgia*. But in the latter the pain starts nearer the median line or to the left of it; there is rarely a chill or fever, there is no jaundice, no calculus in the stools. **Renal colic** has its pain more in the back, and radiating to the thigh, penis, and testicle, and is accompanied or followed by blood or calculus in the urine. The cardinal diagnostic features in hepatic colic are pain in the upper half of the abdomen and to the right of the median line, radiating toward shoulder, vomiting, shock, subsequent jaundice, tenderness over region of gall-bladder, calculi in the stools, history of previous attacks.

CIRRHOSIS OF THE LIVER.

Patients suffering from hepatic cirrhosis are, usually, adult males, the victims of chronic alcoholism or of syphilis. Women, children and infants are more rarely affected. The disease may be the result of a chronic passive congestion, as from heart-disease. The attention is, usually, first attracted to the stomach by the poor appetite, coated tongue, nausea or vomiting. These are the evidences of the gastric catarrh, brought about, largely, by the passive congestion of the gastric portal radicles. Diarrhœa, with pasty stools, and hemorrhoids, may still further show portal congestion. In some cases constipation obtains. Hemorrhages from the stomach or bowels may occur, and be the first symptom to occasion alarm.

Often, in the first stage of cirrhosis, the liver is enlarged so that there may be a complaint of a sense of uneasiness and weight, or even of dragging pain, in the right hypochondrium. As portal obstruction becomes more marked, the gastro-intestinal symptoms grow more pronounced, and abdominal ascites is produced. This may reach an extreme degree. The feet and legs usually become œdematous after there are evidences of fluid in the abdomen. Jaundice is rare, though a muddy-colored skin is commonly seen. This is often itchy. There is loss of flesh and strength. There is, frequently, a diminished amount of urine, and traces of albumin and of sugar are at times found.

The picture of advanced cirrhosis is often strikingly characteristic, the emaciated upper half of the body with its dirty, dry, wrinkled skin, showing in marked contrast to the lower half, with its œdematous legs and protuberant abdomen, over the tightly stretched and glossy skin of which course the tortuous and distended veins.

Splenic enlargement and hepatic contraction can be made out by percussion, though it may be necessary to withdraw the fluid from the abdomen before percussion is satisfactory. Palpation, at times, enables one to feel the hard and slightly roughened liver just under the costal arch. Usually, the edge cannot be felt. In the early stages the enlarged liver can be demonstrated.

Without ascites, or very perceptible decrease in the volume of the liver, the diagnosis may be only presumptive.

In distinction from atrophic cirrhosis, the clinical picture of which has just been given, there is a form known as **hypertrophic cirrhosis**, where there is a permanent enlargement of the organ. In its restricted sense the term is applied to those forms where the connective tissue starts from the periphery of the smaller bile-ducts, instead of from the branches of the portal vein, as is the case in the atrophic form.

In this form the liver remains large, jaundice is marked, ascites and dropsy may be slight. Acute toxic symptoms, as in acute yellow atrophy, may supervene at any time. Delirium, fever, typhoid state, convulsions may all be present. Death usually occurs after these attacks, in a few days or weeks. The liver does not, as in acute yellow atrophy, diminish in size.

CANCER OF THE LIVER.

Cancer of the liver is rarely primary. Sarcoma occurs oftenest in children, carcinoma in adults. Aside from the usual anæmia, weakness, emaciation, cachexia, there is found an enlargement of the liver often, especially late in the disease, causing a distinct bulging in the hypochondriac and epigastric regions. Nodules may be felt over the surface of the enlarged liver. Jaundice is present in fifty per cent. of cases; pain may be moderate or severe. Secondary deposits are common in other organs. A temperature of 100° or 101° is common. Death occurs, usually, within twelve months from the occurrence of symptoms.

ABSCESS OF THE LIVER.

Infection of the hepatic parenchyma with pyogenic organisms may occur in the following ways: 1. Trauma, as from stab or bullet wound. 2. Extension from neighboring organs, as from the gall-bladder, peri-appendicular absce s, perforating gastric ulcer. 3. Through the bile ducts, *i. e.*, from suppurative cholangitis, often the result of cholelithiasis. 4. Through the portal vein, as in cases of dysentery, typhoid fever, enteritis. In tropical climates especially there is a close connection between dysentery, especially the amœbic form, and liver abscess. 5. Through the hepatic artery, as in pyæmia, where septic emboli may reach the liver by this channel. 6. Through the hepatic veins, *i. e.*, by an embolus reaching the liver against the blood current—a rare occurrence. 7. In children, through the umbilical vein.

Males furnish by far the larger number of cases.

The exact origin of some tropical abscesses is still a matter of hypothesis.

These abscesses may be small, or may reach the size of the fœtal head; they are multiple or single, may coalesce or may rupture into the abdominal cavity, or, if a protecting adhesive perihepatitis has preceded, into a neighboring organ or cavity, as the pleural cavity, the colon, the pericarduim, etc.

Symptomatically, we often have the symptoms of the primary affection, as a dysentery, typhoid fever, abscess of the brain, ulcerative endocarditis, gall-stones. In some cases there are stormy symptoms of chills, fever, sweating, local tenderness, enlargement of the liver, all calling attention to this organ as the seat of an inflammation, evidently purulent. In other cases, and, probably, the majority in this climate, the disease is more insidious in its approach. The abscess may remain latent for weeks or months, causing but slight inconvenience. In these cases of a subacute type there is complaint of malaise, deranged appetite, irregularity of the bowels, some pain in the region of the liver, augmented by deep breathing or pressure over the liver, and a gradual failure in strength. Fever is noted toward evening, though the thermometer may not register more than 100°. Sweats are complained of, there may be chills or chilly sensations, and finally the patient comes under the care of the physician, weak, emaciated, with daily temperature and much the appearance of typhoid fever. Probably, the condition is oftener mistaken for typhoid than for any other disease, for as it progresses, delirium, rapid pulse, diarrhœa, etc., may make the resemblance the more striking.

Physical examination in most cases enables a diagnosis to be made, though in small abscesses, especially if located centrally or posteriorly, a diagnosis may be impossible. Locally there is pain on movement of the organ, as by deep inspiration or by deep pressure over it. Pain may be constantly present when there is much accompanying local peritonitis. On percussion and palpation the liver is found enlarged vertically, a line of dulness with its convexity upward, reaching at its highest point anteriorly the fifth rib or even higher, being strongly indicative of abscess. Below, the limits may reach even to the umbilicus. A prominence in the right hypochondrium is not infrequently seen. Œdema over this area, even though slight, is of great diagnostic value. Where fluctuation and redness are present, as sometimes occurs, suppuration is easily recognized. There is at times moderate icterus. The right decubitus is common, and tension of the right rectus abdominis muscle may be noted. Cough (liver cough), dyspnœa, usually show involvement of the diaphragm.

There is a minimum danger following an aseptic exploratory puncture which will clear up many a doubtful case. Multiple small abscesses or a single small one may be missed by the needle.

Differential Diagnosis. **Typhoid Fever.** In abscess, history of preceding bloody diarrhœa or gall-stones, fever more irregular, chills, sweats, less tympany, no rose-spots, bronchitis (bilateral) absent; evidences of local trouble in the hepatic region.

Malarial Fever. History of infection, periodicity, yields to, or are greatly modified by, quinine; plasmodium in the blood.

Pulmonary Tuberculosis beginning in lower right lobe; cough, sputa, tubercle bacilli. Abscess of the liver may cause inflammatory consolidation of the lower right lobe, followed by abscess; may cause right-sided empyema or rupture into a bronchus, and simulate abscess of the lung.

Abscesses originating in tuberculosis of a *rib*, *vertebra*. Careful physical examination or exploratory operation will determine the cause.

Suppurative Pleurisy, with depression of the liver. Here a diagnosis is at times impossible, and abscess of both the liver and pleura may co-exist. The history of the case, the high position of flatness behind, the absence of the convex line of flatness in front, and at times the change of the line of flatness with change in position of the patient, clear up the diagnosis.

DIFFERENTIAL DIAGNOSIS OF ENLARGEMENTS OF THE LIVER.

The main points of value in establishing a differential diagnosis between the different forms of enlargement of the liver are here given:

1. **Chronic Passive Congestion.** Consecutive to valvular heart disease, emphysema. Varies in size from day to day; may be hepatic pulse; jaundice slight.
2. **Cancer.** Adults, usually pain; cachexia and progressive anæmia with leucocytosis; evidence of primary cancer; liver often rough and nodular; temperature may reach 100° or 101°; jaundice common.
3. **Abscess.** History of dysentery, diarrhœa, suppurating hemorrhoids, gall-stones; pain, irregular fever, chills, and sweats; liver smooth, usually enlarged vertically; may be redness or fluctuation; jaundice usually slight; pus by aspiration; leucocytosis, peptonuria.
4. **Echinococcus.** Slow development, often painless; frequently of great size, with little or no impairment of general health, the symptoms those of pressure; fremitus or "hydatid thrill;" fluid by aspiration is usually light yellow, non-albuminous, saccharine, and may contain hooklets.
5. **Syphilis.** Often not to be distinguished from ordinary cirrhosis; usually painful; gummatous nodules may be felt; history of syphilis or evidences, as scars, of the disease; liver may diminish in size under iodides.
6. **Hypertrophic or Biliary Cirrhosis.** Jaundice early and marked; liver often large for one or two years; may be sudden deepening of jaundice and symptoms of cholæmia, death following in few days; ascites uncommon.
7. **Fatty Liver.** Excess of fat in other parts of body; patient a chronic alcoholic or sufferer from tuberculosis; liver smooth, hard; edge perhaps slightly rounded.
8. **Amyloid Liver.** History of syphilis or evidence of tuberculosis, cancer, suppuration; usually spleen, kidney, or intestines are amyloid; liver smooth, may reach to umbilicus.
9. **Enlarged Liver of Malaria and of Leukæmia** recognized by blood examination.

ACUTE YELLOW ATROPHY OF THE LIVER.

Acute yellow atrophy may begin with a sudden vicious onset or be ushered in by prodromal symptoms of anorexia, nausea, diarrhœa, malaise, slight fever, these symptoms lasting for a few days or even a few weeks. The occurrence of jaundice after such prodromata may lead to the diagnosis of catarrhal jaundice. Severe nervous symptoms, however, call attention to the gravity of the disorder. Headache is agonizing. The patient grows restless, sleepless, wildly delirious, later sinking into a condition of semi-stupor, lapsing into coma. Muscular twitchings, or rarely general convulsions, are met with. The icterus becomes more marked, the urine shows bile pigment, leucin, and tyrosin crystals, decreased amount of urea. Vomiting may be persistent, constipation or diarrhœa may be present, subcutaneous or submucous hemorrhages, epistaxis, hæmatemeis, melæna may occur.

There is noted about some of these patients a peculiar sweetish odor. The temperature, usually slightly elevated, may be normal or subnormal. Toward the end it is commonly quite high. The pulse, at first slow, later becomes very rapid. Da Costa lays stress upon the fact that there are sudden and unaccountable variations in the pulse rate, it being at one time slow and again very rapid.

A slight increase in the size of the liver is soon followed by a rapid diminution in size. The organ may be reduced to one-third its normal volume.

Phosphorus poisoning furnishes a clinical picture very similar to acute yellow atrophy. In the former disease there is no leucin or tyrosin in the urine, the liver is more painful, is longer enlarged, and the maniacal excitement is less marked.

Where prodromata last for several days and the patient passes into the typhoid state, *typhoid fever* may be diagnosed, and especially as in acute yellow atrophy, there is, as in typhoid, splenic enlargement. The course of the pulse and temperature, the convulsions and wild delirium, the gradually deepening jaundice, leucin and tyrosin, and rapidly diminishing liver, enable one to make the diagnosis late, though it may early be impossible.

Acute yellow atrophy is found oftenest in women, and very frequently associated with pregnancy or parturition.

PYLETHROMBOSIS. CHRONIC ADHESIVE PYLEPHLEBITIS.

Thrombosis of the portal vein can rarely be diagnosed. The sudden appearance of portal engorgement, ascites, enlarged spleen, intestinal and gastric catarrh, during cirrhosis of the liver or when pressure upon the vein can be suspected, as by a cancer, aneurism, inflammatory bands, enlarged liver, points strongly toward thrombotic obstruction of the vein. In case collateral circulation is established there may be a later disappearance of evidences of portal engorgement.

SUPPURATIVE PYLEPHLEBITIS.

Suppurative pylephlebitis is secondary to suppuration in the neighborhood of the vein or its radicles, some of which become involved, or it is part of a general pyæmia. Appendicitis is perhaps the commonest cause. The symptoms are those of pyæmia, with an enlarged, tender liver the same as in abscess. Jaundice is common. Death is seldom delayed longer than four weeks. A knowledge of the primary disease is of great aid in diagnosis.

DISEASES OF THE PANCREAS.

It is only recently that the literature of pancreatic disease has become full enough to warrant the description of clinical phenomena definite enough to be of diagnostic value. Fitz, Senn, Seitz, among others, have added much to the knowledge of these obscure affections, even now but imperfectly understood.

HEMORRHAGE INTO THE PANCREAS.

Rupture of a bloodvessel of the pancreas, with resulting infiltration of the organ, causes shock and death, through the solar plexus. This hemorrhage, while occurring by preference in patients with atheromatous vessels, may take place in the apparently perfectly healthy, and may be unheralded. The pain is sudden, severe, located in the upper zone of the abdomen. Nausea and vomiting are early and persistent features. The evidences of shock are marked, the pulse becomes rapid and weak, the surface cool, the patient restless, the temperature often subnormal; constipation and tympany are the rule. Collapse soon supervenes, and death occurs in from a few minutes to several hours, or even days, from the onset of pain. The most striking symptoms are "pain, vomiting, anxiousness, restlessness, and the state of collapse into which the patient soon falls."

ACUTE PANCREATITIS.

Acute pancreatitis presents many of the symptoms of intestinal obstruction, or of perforative peritonitis from duodenal or gastric ulcer. The severe pain which comes on without warning is referred to the epigastrium. Nausea, vomiting, constipation, tympany soon follow, and in almost all cases death ensues in from one day to four days, the patient often rapidly going into collapse. Osler records a case of recovery. Temperature in acute pancreatitis may be normal or slightly elevated.

Pancreatic gangrene is followed by peritonitis and death.

Seitz,[1] in the conclusion of his exhaustive article, after referring to the fact that, since attention has been directed to the pancreas as the possible seat of hemorrhage or gangrene, the so far small number of ante-mortem diagnoses will be increased, gives the following as the symptom-complex in typical cases: "Without warning, in the midst of health, a sudden, severe attack of pain in the region of the stomach or its immediate neighborhood, vomiting, collapse, swift death, before there is pronounced peritonitis, in a few minutes, a few hours, or a few days. Early manhood, obesity, syphilis, atheroma, old age—all can act as favoring circumstances. There has occurred a rupture of a bloodvessel, either in the pancreas or its vicinity, from this a hemorrhage, fatal through its attack upon the nerve plexuses in this region."

CANCER OF THE PANCREAS.

Cancer of the pancreas, usually involving the head of the organ, can in some cases be palpated deep in the epigastric region. Jaundice is an almost inevitable result of tumor of the head of the pancreas, as the ductus communis choledochus is obstructed by pressure or direct invasion by the new growth. Secondary deposits in the liver and stomach may add the symptoms of the secondary disease to that of the pancreatic affection. Fatty stools are commonly met with where the duct of Wirsung is occluded.

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[1] Zeitschrift für klinische Medicine, 1892, Bd. xx., S. 328.

The symptoms would therefore be epigastric pain, perhaps swelling and a fixed tumor felt on palpation, fatty stools, almost invariably jaundice, cancerous cachexia, increasing anæmia, leucocytosis.

CYST OF THE PANCREAS.

Cyst of the pancreas may be slow or rapid in developing. The swelling first noted in the epigastrium may fill the abdominal cavity, simulating an ovarian or echinococcus cyst. Pain and cachexia are lacking. Aspiration shows a clear serous fluid, though oftener with a brownish color.

Exploratory laparotomy has been found necessary in many cases of pancreatic disease in order to establish a positive diagnosis. A positive conclusion can only be reached by exclusion.

DISEASES OF THE PERITONEUM.

PERITONITIS.

Acute general peritonitis commonly arises from infection spreading from some neighboring organ or tissue covered by peritoneum. The uterine lymphatics and the Fallopian tubes furnish a common avenue of microbic entrance. Perforation of any portion of the alimentary canal allows bacteria and products of digestion to escape into the abdominal cavity. In this way appendicitis is a common cause of peritonitis, as may be, by perforating, an ulcer, typhoid, stercoral, tubercular, or peptic. Suppurative inflammation may, by direct extension or by rupture of an abscess, produce suppurative peritonitis. Pyosalpinx, appendicular abscess, empyema of the gall bladder, etc., are examples of the original cause of many cases.

Very rarely peritonitis seems to develop during acute rheumatism or chronic nephritis, involving the serous covering of the intestines in a manner apparently analogous to that in which the other serous membranes, the pleura, and endocardium are attacked. Local peritonitis, plastic or adhesive, often occurs where there is inflammation in the underlying tissue. Thus we have adhesive peritonitis about a salpingitis, about a splenic infarct, an abscess of the liver (perihepatitis) or over an intestinal or gastric ulcer.

Pain, except in those profoundly septic cases where the intoxication is so extreme as to depress the sensory centres beyond the point of recognizing pain, is excruciating. It is important early to locate the site of the initial pain, as it may be the only clew to the original cause of the difficulty, for soon the pain is diffuse over the entire abdomen. Tenderness to pressure is extreme, the patient not only trying to ward off the examining finger, but complaining even of the weight of the bed-clothes. Involuntarily, too, he aims to avoid moving the abdominal muscles, by flexing the knees and thighs, by lying quietly on his back, and by ceasing abdominal breathing. Breathing is therefore of the thoracic type, and often somewhat rapid. Opium, so commonly given for the relief of pain, may mask the symptoms so that comparatively slight pain is complained of, the patient moves quite freely in bed, and the breathing is slow.

With the initial pain, which is often sudden and unheralded by premonitory symptoms, there is frequently a chill, and soon there are manifested evidences of gastro-intestinal disturbance. Vomiting comes on early and is often continuous. Rarely it is stercoraceous; oftener of a grass-green color. Hiccough may, later, replace the vomiting. Constipation is marked from intestinal paresis, and for the same reason tympany is extreme, often pressing upward the diaphragm, liver, and heart. The apex-beat may be found in the fourth space. There is no rule as to fever. Commonly the temperature ranges from 101° to 104°, though in cases, and these some of the severest, the temperature may be low. The pulse is small, quick, rapid, wiry. Great prostration and weakness come on early. The face betrays the evidences of suffering, the nose is pinched and cold, the lips cyanosed, and the eyes sunken. The Hippocratic countenance is common. The mind is at first unclouded, though later delirium may be prominent. Indicanuria is common.

When a large amount of fluid is in the abdomen dulness may be found in the lowest portion of the abdomen. Usually tympany is so extreme as to conceal any evidences of fluid. Liver dulness may be lessened by the pushing back of the liver from the anterior wall of the abdomen by a distended coil of intestine, or it may disappear from the presence of a large amount of gas loose in the abdominal cavity, as after a typhoid perforation.

The exploring needle as a means of diagnosis is to be discarded in favor of exploratory incision in doubtful cases, or where the question of surgical interference comes up.

Localized peritonitis gives the same symptoms as the diffuse, save that the pain is more limited to one spot, and the other symptoms mentioned are less in degree. Vomiting, constipation, and tympany may be absent or slight. Constipation is the rule. There is much less evidence of shock and collapse, and the tendency is, if the inflammation be strictly localized, *i. e.*, shut off from the general cavity by adhesions, as in the case of a pelvic peritonitis, subdiaphragmatic empyema, peri-appendicular abscess, for many of the symptoms gradually to disappear, and the only remaining evidence of the disease to be such as would come from peritoneal adhesions or an encapsulated collection of pus.

General peritonitis is rarely mistaken for any other disease. The mistake is oftener made of taking some other affection for peritonitis, as gastralgia, gall-stones, intestinal obstruction. The points in differential diagnosis are given under the headings of the other diseases. It is of the utmost importance that the physician strive to make an early diagnosis of peritonitis, and one including the causation of the disease, as surgical aid promptly given in these cases often saves a life that would otherwise be lost.

CHRONIC PERITONITIS.

Chronic peritonitis may be of the adhesive type, local or general; it may result in great thickening of the peritoneum and the development of a large amount of connective tissue, causing contractions and deformities. Tuberculosis or carcinoma of the peritoneum lead to a chronic form of inflammation.

Diagnosis of this affection is often made only post mortem, as no symptoms during life attract attention. Pain is variable; friction sounds uncertain. Ascites may be of moderate or extreme degree, the exudate in tubercular or cancerous affections being frequently hemorrhagic. Bands of adhesions may cause pain or interfere, mechanically, with the functions of various organs. A thickened omentum, as in tuberculosis or carcinoma, can at times be made out. The existence of primary tubercular or carcinomatous deposits, the gradual development of cachexia, may give evidence of the true nature of the peritonitis.

ASCITES.

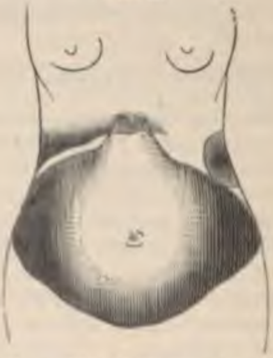
The secret of the successful diagnosis of ascites, which rests almost entirely on the physical examination, lies in remembering that the fluid, loose in the abdominal cavity, sinks to the lowest portion in whatever position the patient is placed.

Inspection shows a uniformly bulging contour most prominent, in the erect position, in the lower portion of the abdomen. As the patient lies upon the back there is a flattening of the central portion of the abdomen, with a bulging laterally, unless there is an excessive amount of fluid, when no change is noted. It is important to remember this point, as it is in contrast to the preserved prominence in the centre, when an ovarian cyst bulges prominently forward as the patient assumes the dorsal position.

The embarrassed respiration, shiny skin, protruding navel, distended veins, can be noted as well as the displacement upward of the apex-beat of the heart.

Palpation imparts the yielding sensation of movable fluid, and if the abdomen, on the side opposite the palpating hand, be given a sharp tap, the wave-like impulse is felt against the palpating hand (fluctuation).

FIG. 22.



The shading indicates the position of the *percussion-dulness in a case of ascites*, while the patient is lying on the back, the fluid falling to the low levels in the flanks, and the umbilical region remaining clear. (FINLAYSON.)

Percussion should be practised in at least two different positions, in order to note the variations in the line of dulness.

The intestines, unless held by adhesions, float always in the uppermost zone of the abdomen, so that in whatever position the body may be, we have flatness below and resonance above. In the erect posture, for example, the line of dulness is a straight line across the abdomen, its height dependent on the amount of fluid. In the dorsal position there is a central resonant area, surrounded by a zone of dulness. In ovarian cyst the exact opposite obtains, dulness occupying the centre, which is encircled by resonance.

Auscultation is negative. Exploratory puncture, with a fine needle, should be employed in all doubtful cases, and always before operative aspiration. This would avoid such mistakes (and these have occurred in the hands of good men) as puncturing the bladder, the pregnant uterus, a cyst of the ovary, uterine fibroid, or even the intestine of intestinal obstruction distended with gas and liquid. The character of the fluid is also determined by this exploration, and may be of importance in determining the character of the affection causing the ascites, for the latter is always to be looked upon as the result of some antecedent disease. Thus, milky, chylous fluid—chylous ascites—points, usually, to obstruction of the thoracic duct; fluid rich in blood is oftenest found in carcinomatous or tuberculous affections of the peritoneum. Tubercle bacilli are at times found in fluid from cases of tuberculosis of the peritoneum. A transudate is usually lighter in color, of lower specific gravity (under 1018), less rich in albumin and formed elements, less rapidly coagulable than an exudate.

Chyle has practically the composition of that fluid as found in the lacteals, and always shows a distinct trace of sugar (Senator). Certain milky fluids, sometimes found in connection with chronic carcinomatous or tuberculous peritonitis, and due to fatty degeneration of cells and not to genuine transudation of chyle, are spoken of as chyliform, the affection as **chyliform** or **adipose ascites.** Exploratory puncture affords the only means of recognizing the chylous or chyliform character of ascitic fluid. In the chyliform ascites fat is abundant, extractable by ether; there is no sugar. The **filaria sanguinis hominis** has been found in a few cases of chylous ascites.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE AND LARYNX.

ACUTE RHINITIS. CORYZA.

Exposure to cold, udden atmospheric changes, the inhalation of irritating gases, in many cases microbic influences, may produce an acute inflammation of the nasal mucosa often spoken of as coryza or a "cold in the head." Constitutional disturbances, as fever, headache, backache, malaise, anorexia, may be slight or fairly well marked. Chilly sensations are not uncommon at the commencement of the attack. The nasal mucous membrane becomes swollen, interfering with nasal respiration, and soon there is a thin, watery, often irritating discharge, which keeps the patient almost constantly "sniffing" and using the handkerchief. The eyes are often watery, the throat somewhat sore, and partial deafness may come on from involvement of the Eustachian tube. The swelling subsides in a few days, though a muco-purulent nasal discharge may continue for some time after.

No difficulty attends the diagnosis of acute rhinitis, though we always have to keep in mind that it may be but a symptom of measles, epidemic influenza, or hay fever.

CHRONIC RHINITIS.

A form of chronic rhinitis, usually termed simple rhinitis, occurs in which there are painful spots on the mucosa, detected by the probe, redness and some swelling, and more or less constant discharge. Acute coryza easily sets in. The continuance of this form readily leads to hypertrophy of the mucous membrane, especially that covering the turbinated bones and the septum. Difficulty in nasal breathing, nasal twang to the voice, dropping of mucus into the pharynx, perhaps deafness from a coincident naso-pharyngeal catarrh, are the symptoms of this affection. The swelling and redness, on examination, render the diagnosis easy.

An **atrophic** form of rhinitis occurs in which the destruction of the membrane leaves an enlarged nasal cavity. Profuse discharge, crusts, offensive odor enable one to make the diagnosis. The offensive odor may be present without actual ulceration or necrosis of bone. The patient's anosmia prevents his detecting the foul smell.

HAY FEVER.

This resembles the ordinary acute rhinitis, but is recurrent, usually appearing about the middle of August in susceptible persons. Sneezing is frequent, the nose runs, the eyes are watery, there may be cough and asthmatic phenomena. The nasal mucous membrane is greatly reddened, swollen, and in places hyperæsthetic. The severity of the attack, the time of its occurrence, and its return at the same time year after year, with its disappearance with the first frost, enable a diagnosis to be made.

ACUTE CATARRHAL LARYNGITIS.

The diagnosis of this affection is made by its frequent association with pharyngitis and rhinitis, the dry cough, sense of tickling and of pain in the larynx, the husky or whispering voice. Laryngoscopic examination shows swollen, lustreless, and reddened vocal cords, often with mucus covering them, and perhaps stretching between them in thread-like bands. The other laryngeal structures, notably the aryepiglottidean folds, are usually more or less swollen.

A slight grade of fever, which is often present, may aid in distinguishing this disease from hysterical aphonia and simple laryngismus. Laryngoscopic examination will settle the diagnosis in the latter case.

The constitutional and faucial symptoms, together with the examination by the laryngoscope, where this is possible, serve to separate diphtheritic laryngitis from the simple affection.

CHRONIC LARYNGITIS.

Chronic laryngitis may be suspected when repeated acute attacks are followed by an almost constant desire to cough, a tickling referred to the larynx, and an alteration in the voice. The laryngeal mucous membrane, on examination, is found swollen and slightly reddened.

MEMBRANOUS LARYNGITIS OR CROUP.

Nearly all, if not all, cases of membranous laryngitis are diphtheritic in character, and associated with evidences of the disease in throat or nose. In some, however, the faucial evidences are lacking, the membrane forms primarily upon the larynx, and there are but slight constitutional disturbances. The symptoms, aside from the general symptoms of septic intoxication, are identical in the specific and the supposedly non-diphtheritic forms.

The voice becomes husky, there is an irritating cough, the symptoms are worse at night, but gradually grow more marked, until breathing becomes extremely difficult. There is a wheezing or raucous inspiration and expiration, dyspnœa is marked, the soft parts of the thorax retract instead of expanding during inspiration, and the livid countenance, feeble, rapid pulse, and restless tossing of the child show the imperfect oxygenation of the blood. Membrane may be seen as it is coughed up, or, if it is possible to use the mirror, with the laryngoscope.

From the spasmodic closures of the larynx the membranous form is distinguished by its *progressive* character. It is slower in onset, and there is not experienced any sudden relief unless membrane is coughed up, which fact alone establishes the diagnosis. In **laryngismus stridulus** a child perfectly healthy during the day or night may suddenly, especially if angered, "hold the breath." The veins grow turgid, the lips cyanotic, strangulation seems imminent, when the spasm relaxes and a deep inspiration resembling the whoop of pertussis marks the end of the attack.

Children suffering from a trifling and perhaps overlooked catarrhal laryngitis will awaken at night with a hoarse, ringing cough and intense dyspnœa. Inspiration is slowly performed and is "crowing" in character. They seem about to suffocate, yet rarely does death occur in the attack. Usually, the laryngeal spasm passes away, the child falls asleep, and next morning seems in perfect health, only perhaps to have a repetition of the spasm during the night. This is the common **spasmodic croup.**.

ŒDEMA OF THE GLOTTIS.

Œdema of the glottis may come on during an acute catarrhal laryngitis, or, in fact, as a consequence of any form of inflammation of the larynx. It is also occasinally met with as a complication of nephritis, either acute or chronic. It often develops suddenly. There is difficulty in breathing, the same restlessness, anxiety, and signs of poor aëration of blood that are found in membranous laryngitis. The epiglottis and the laryngeal tissues are seen by the laryngoscope to be greatly swollen, and, perhaps, almost transparent. In the absence of the laryngoscope, or of a proper light, the finger can usually make out the swollen epiglottis and aryepiglottic folds.

Syphilis, tuberculosis, carcinoma may attack the larynx. For the diagnosis of these conditions, usually associated with evidences of the disease in other organs, as well as for the diagnosis of the various forms of laryngeal paralysis, the student is referred to special works upon the diseases of the larynx.

DISEASES OF THE BRONCHI AND LUNGS.

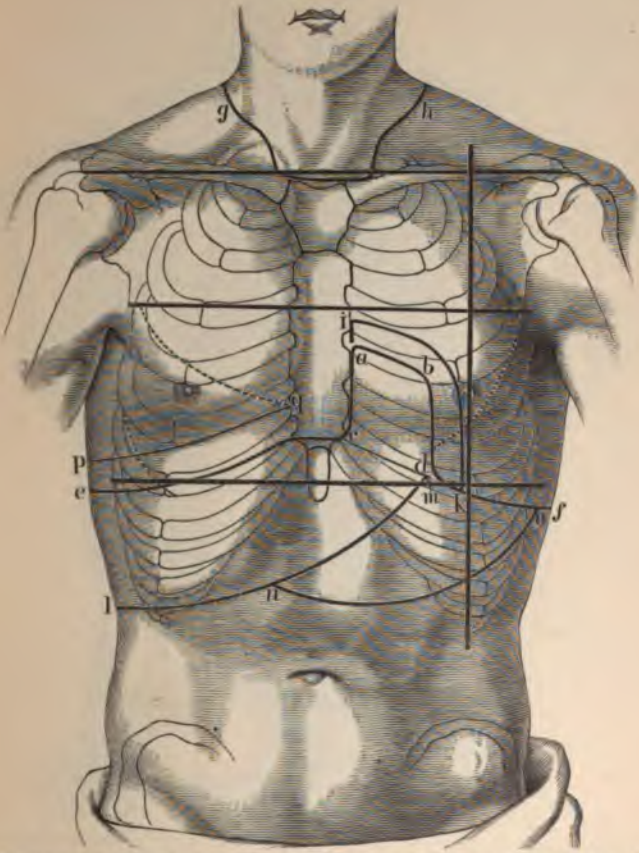
GENERAL CONSIDERATIONS.

The recognition of thoracic diseases depends so largely upon the physical examination that a brief outline of physical diagnosis, as applied to the chest, will here be given.

For purposes of description, the chest wall is divided, arbitrarily, into certain regions. Anteriorly, the region above the clavicle, on either side, is known as the supra-clavicular region, that immediately over the clavicle as the clavicular region, the space between the clavicle and the third rib as the infra-clavicular, between the third rib and the sixth the mammary, and below the sixth rib the infra-mammary. The space above the sternum is called the supra-sternal space, the sternal region proper being divided by the line of the third rib into an upper and lower space. An imaginary vertical line, passing through the nipple, is spoken of as the mammillary line. The lateral region is divided into axillary and infra-axillary regions by a horizontal line, extending from the lower border of the mammary region, *i. e.*, the sixth rib.

Posteriorly, the space occupied by the scapula is known as

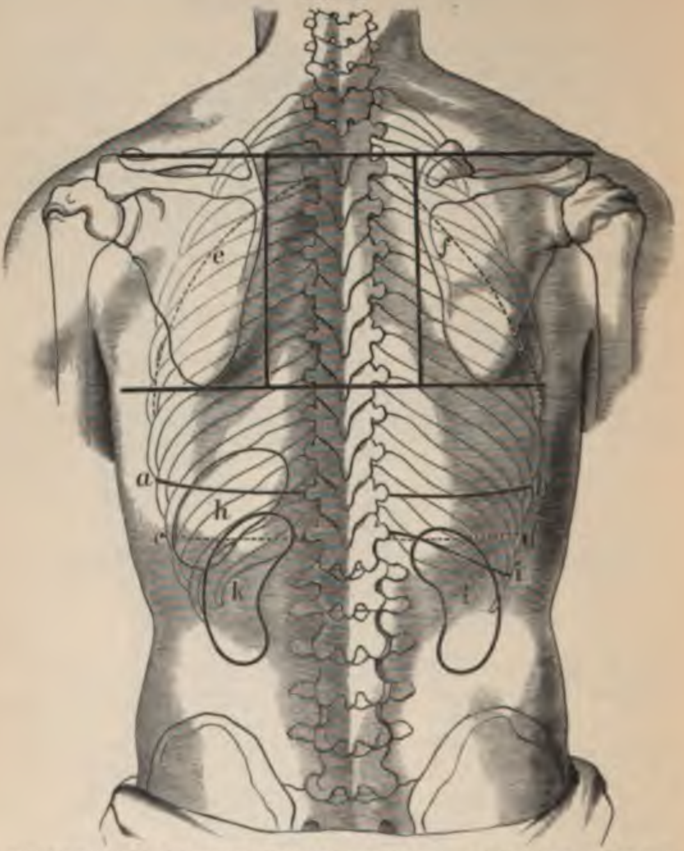
FIG. 23.



The horizontal lines indicate the boundaries of the regional divisions on the anterior aspect of the chest. The vertical line is the linea mammillaris. The oblique dotted lines indicate the interlobar fissures. *a b*, *a c*, *c d* and *b d*, boundaries of superficial cardiac space; *i k*, outer boundary of deep cardiac space; *ce*, lower boundary of right lung; *d f*, lower boundary of left lung; *g h*, upper boundary of right and left lung; *l m*, lower boundary of hepatic flatness; *p q*, upper boundary of hepatic dulness; *n o*, lower boundary of stomach, moderately distended.

the scapular region. The spinous ridge divides this space into upper and lower scapular spaces, or a supra-spinous and an infra-spinous space. All below the level of the lower angle of

FIG. 24.

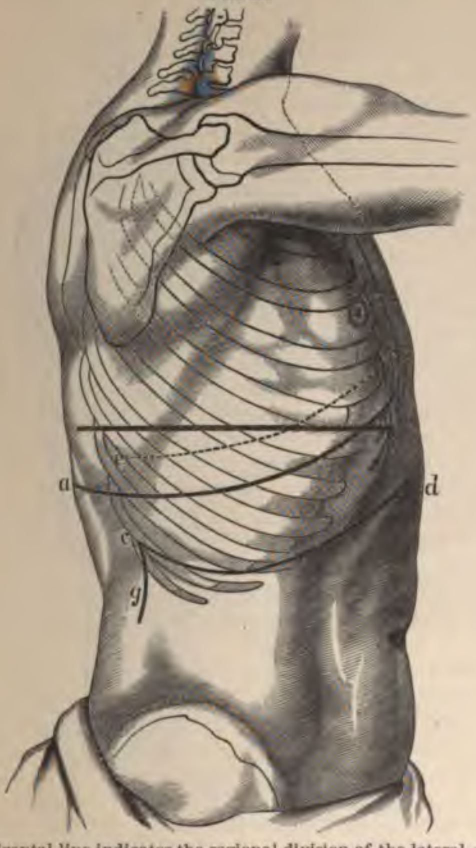


The longitudinal and vertical lines indicate the regional divisions on the posterior aspect of the chest. *a b*, lower boundary of lungs; *c d*, lower limit of expansion of lungs; *e f*, interlobar fissures; *h*, spleen; *i*, lower boundary of liver; *k*, left kidney; *l*, right kidney.

the scapula belongs to the infra-scapular region. The space between the vertebral border of the scapula and the spinal column is the inter-scapular region. The patient is assumed to be erect, with the arms hanging at the sides.

The figures show these divisions of the chest wall and the location of the inter-lobar fissures. The figures are those of

FIG. 25.



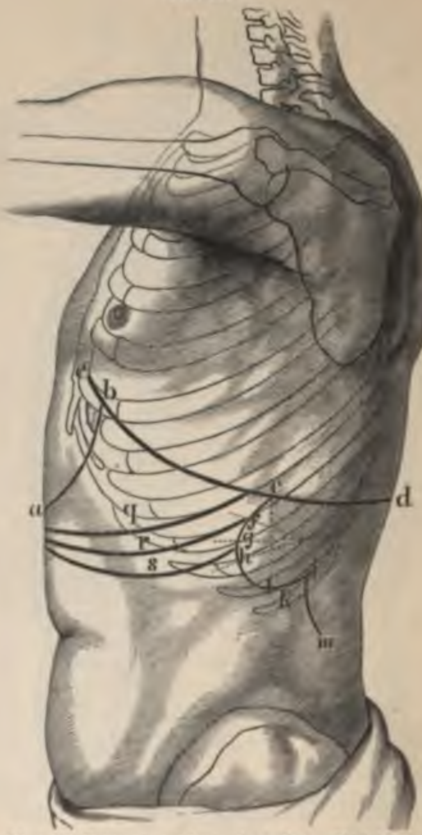
The horizontal line indicates the regional division of the lateral aspect of the chest. *a b*, lower boundary of right lung; *c d*, lower boundary of hepatic flatness; *e f*, upper boundary of hepatic dulness; *g*, border of kidney.

Weil, as modified by Wilson in his edition of Flint's *Auscultation and Percussion.*

PHYSICAL EXAMINATIONS.

The methods of physical examination, as applied to the chest, are **inspection, palpation, percussion, ausculation, mensuration, succussion, exploratory puncture.**

FIG. 26.



ab, boundary of hepatic flatness; *cd*, lower boundary of left lung; *e*, *f*, *g*, *h*, *i*, *k*, *l*, boundaries of spleen; *lm*, boundary of kidney; *q*, *r*, *s*, lower boundaries of the stomach in different degrees of distention.

Inspection is probably the one method most frequently overlooked by the beginner. It is indispensable to a thorough examination that the patient be stripped to the waist. He should sit in a chair, erect, facing the examiner, the arms by the side, the hands upon the knees. The facies of the patient, the muscular development, the presence of œdema are observed. The shape of the chest is to be noted as well as its movements, their rapidity and regularity.

The phthisical chest, with its flat appearance, its sunken infra-clavicular regions, perhaps with unequal depressions on the two sides and unilateral loss of motion, is in striking contrast to the rounded, barrel-shaped chest, with its up-and-down movement, of emphysema. The condition of the intercostal spaces may be of value, their bulging denoting fluid or gas in the pleural cavity. Irregularities of contour, as from rickets, new growths, aneurismal protrusions, will of course be noted. The student should remember that the results of physical exploration are usually obtained by the comparison of the one side with the other. The position of the heart's apex-beat and its area of diffusion can often be made out.

Not only is the chest to be subjected to the inspector's scrutiny, but at the same time the neck and upper abdomen are investigated for confirmatory evidences of thoracic disease. Prominent cervical muscles with dusky countenance, prominent right hypochondrium, epigastric pulsation, may give evidence of emphysema; pulsating arteries or veins may lead to the recognition of valvular disease. Light may be thrown upon an otherwise obscure case by the detection during the thoracic inspection of scars, vertebral deformity, the enlarged breasts with pigmented areolæ of pregnancy, or the pigmentation of Addison's disease. A skilled diagnostician who has carefully trained himself to the thorough practice of inspection finds it one of the most valuable of his methods of examination.

Palpation is the examination by the application of the hands. The sense of touch is here exclusively used. Points of tenderness, irregularities of contour, the bulging of intercostal spaces, the resistance met with on pressure over different areas of the chest surface are noted. The position of the apex-beat is determined, and this, by the laying of the entire hand flat over the precordial region, and not by attempting to locate it with the tips of the fingers. Inequality in the movement of the two sides may denote pleuritic effusion, or, if it is one apex that is motionless, tuberculosis. When both hands are placed on the chest, one on either side, and the patient directed to talk, a thrill or fremitus is communicated to the hands, known as the **vocal fremitus**. Fluid, as in pleuritic effusion or empyema, hinders the ready conduction of the sound-waves, and so lessens vocal fremitus on the affected side. Consolidated lung, on the contrary, providing the bronchi are patent, readily acts as a conductor of the vibration, and fremitus is increased over the consolidated area. Vocal fremitus is of value only when relatively increased or diminished, as the variations in the healthy are so great that in the chest of one person fremitus may be feeble, in another marked. Palpation, aside from fremitus, is rather a means of confirming the results of inspection as to loss of motion, apex-beat, etc., and of auscultation, for the same cause that produces cardiac murmurs, serous friction sounds and bronchial râles often communicates a thrill to the palpating hand.

Mensuration may be spoken of here, as it is useful to determine by actual measurements the size of either side of the chest, the expansile capacity of the chest as a whole and of each half of the chest. It is a confirmatory test of the results obtained by palpation and inspection.

Percussion is best performed by striking a succession of quick, gentle blows, vertically delivered, with the tip of the middle finger of the right hand upon the middle finger of the left hand, which is laid evenly and with gentle pressure upon the portion of the chest wall to be examined. Hammers and pleximeters may be substituted for the fingers. Corresponding parts of the chest on the two sides are to be examined, and the sounds elicited compared, for there is no absolute, normal standand of resonance to which all chests conform. "Speaking broadly, each person furnishes his own standard."

A percussion sound is **resonant** when there is air contained in the part over which percussion is made, **flat** when there is no air, **dull** when the resonance is diminished, *i. e.*, approaches flatness. Dulness is a relative term, and we often speak of slight dulness, marked dulness, absolute dulness or flatness. A good place to note these differences is over the right chest, where above there is resonance which merges into dulness and flatness as percussion is made from above downward and the region of the liver is reached. Resonance varies in **intensity, pitch, quality.**

By **intensiy** is meant the **loudness** of the note, this depending largely upon the force of the blow. Thin, elastic walls and a large amount of air also increase the intensity.

Pitch is relatively low over normal lung tissue. Over a solid it is high, as it is over a large air-containing but non-vesicular cavity, as the stomach or intestine, where there is a tympanitic resonance. Abnormal conditions alter, therefore, the vesicular resonance by raising its pitch.

Quality is the peculiar character of the sound, giving it its distinctive mark, indescribable in words and only recognized by practice. Over air-vesicles the quality is **vesicular.** Over a large body of air, as in the stomach, the quality is tympanitic. A mixture of the two, as in emphysema, is **vesiculo-tympanitic.** "Any resonance which is non-vesicular is tympanitic." (Flint.) The duration of a note of low pitch is longer than that of one of high pitch.

The **sense of resistance** to the finger becomes, by training, of value in diagnosis, fluids or solids furnishing the greater resistance. In children, particularly, the sense of resistance may enable the examiner to recognize by percussion alone that the flatness is due to fluid, and not to a solid or a consolidated lung. The "feel" or the sensation communicated to the examining finger is that of fluid, slightly yielding under the percussion stroke, and not that of an unyielding solid.

The varieties of resonance, therefore, are:

1. **Flatness** or absence of resonance, signifying fluid in the pleural cavity or in a pulmonary cavity (hydrothorax, empyema), fluid in the vesicles (œdema, infarct), solidification of lung (pneumonia, tuberculosis), tumors or abnormal enlargement of structures within the chest (cancer, aneurism).

2. **Dulness** or diminished resonance—due to an abnormal proportion of solids or liquids over the air in the pulmonary vesicles. The same causes that produce flatness, when operating in a slighter degree, produce dulness.

3. **Tympanitic resonance** where there is a resonant but non-vesicular tone, the intensity being extremely variable, the pitch, with few exceptions, elevated. It is met with over air-containing but non-vesicular cavities, as in pneumothorax, pulmonary cavities, over the bronchi and lower trachea, especially noticeable in solidification of the upper lobe and in some cases of bronchiectasis. A tympanitic note is sometimes conducted over the chest from the stomach or inflated colon.

4. **Vesiculo-tympanitic.** A mixture of the vesicular and tympanitic, as in emphysema.

5. **The cracked-pot resonance.** If the hands are clasped palm to palm, leaving an air-space between them which communicates in one direction with the outside air, and if then the back of the underhand be struck forcibly against the knee, the air is forced through the opening of communication with the outside, and produces a sound resembling the cracked-pot sound, like that made by striking a cracked metallic vessel. In the lungs a cavity with lax walls communicating with a bronchus gives forth on percussion the cracked-pot sound.

6. Amphoric resonance. An air-containing cavity with rigid walls and communication with a bronchus gives rise, on percussion, to the metallic-sounding, amphoric resonance, such as is obtained by striking on an empty cask or by percussing the cheek distended with air.

Auscultation is **immediate** when there is no instrument employed, the ear of the listener being applied directly to the chest; **mediate** when some instrument, preferably a binaural stethoscope, is used as an aid. Two types of respiratory sound are heard on listening over the normal chest, the one bronchial breathing, exemplified by tracheal respiratory sound, and the other vesicular, heard most characteristically over the lower lung behind. **Bronchial respiration** may be said to be of marked intensity, high pitch, harsh or tubular quality, and with the expiratory portion separated by a slight pause from the inspiratory, and of longer duration and higher pitch. The further removed from the trachea and larger tubes we go the more the respiratory sounds lose their bronchial character and acquire a vesicular element, until from a bronchial respiration we have a broncho-vesicular tone and then a pure **vesicular murmur.** This murmur is of varying intensity: loud in children, in the old or weak perhaps scarcely audible; it is of low pitch, with a faintly blowing expiratory tone, lower pitched and of much shorter duration than the inspiratory, and of an unique quality, variously described as soft, murmuring, breezy, or vesicular.

The **voice sounds** vary in different chests and in different parts of the same chest, being more intense and concentrated the nearer we approach the bronchi, though spoken words are rarely understood. The whispered voice can scarcely be made out in a normal chest, save over the bronchi or at the apex.

The following table, based upon Flint's classification, shows the sounds heard in diseases of the respiratory organs:

A. MODIFICATIONS OF NORMAL RESPIRATORY SOUNDS AS REGARDS

1. *Intensity.*	
1. Increased vesicular murmur *e.g.*	Vicarious emphysema.
2. Diminished vesicular murmur .	Vesicular emphysema.
3. Suppressed respiratory sound . .	Hydrothorax.
2. *Pitch and Quality.*	
1. Bronchial respiration	Pneumonia (second stage).
2. Broncho-vesicular respiration .	Beginning tubercular consolidation.
3. Cavernous (where walls collapse)	Late stages of tuberculosis.
a. Broncho-cavernous	" " " "
b. Vesiculo-cavernous	" " " "
4. Amphoric respiration	Pneumothorax.

3. *Rhythm.*	
1. Shortened inspiration	Vesicular emphysema.
2. Prolonged expiration	" "
3. Interrupted inspiration	Pleurisy (with pain).
B. ADVENTITIOUS RESPIRATORY SOUNDS OR RALES:	
1. *Laryngeal and Tracheal.*	
1. Dry	Spasmodic croup.
2. Moist	"Death-rattle."
2. *Bronchial.*	
1. Moist.	
a. Large or coarse	Œdema of lungs.
b. Small, fine, or subcrepitant .	Capillary bronchitis.
2. Dry.	
a. Coarse or sonorous	Asthma.
b. Fine or sibilant	"
3. *Vesicular* or *crepitant*	Pneumonia (first stage).
4. *Cavernous* or *gurgling*	Late tuberculosis.
5. *Pleural.*	
1. Friction sounds	Acute pleurisy.
2. Metallic tinkling	Pneumothorax.
3. Succussion sounds	Pneumohydrothorax.
6. *Indeterminate*—crackling sounds . .	Early tuberculosis.

A few words of explanation of some of these sounds are here added.

An increased vesicular murmur, as in vicarious emphysema, is known as **supplementary**. It is **puerile**, because normally found in children. An increased murmur is distinguished from bronchial breathing by the absence of prolonged and high-pitched expiratory sound. A vesicular murmur, normal as regards quality, rhythm, and pitch, may be **feeble** from partial occlusion of bronchi, from an overlying pleural exudate or thickening, by restricted movement of one side, as in paralysis, acute pleurisy, intercostal neuralgia where pain causes the involuntary restriction of motion. Complete **absence or suppression** of the vesicular murmur is met with where no air enters the alveoli, as in obstruction of a bronchus or where a new growth, fluid in the pleural cavity, an aneurism, crowd out the air or interpose so thick a solid layer between the air and listening ear that auscultation fails to discover any sound.

Bronchial respiration has a high, tubular, inspiratory sound of varying intensity, an expiratory sound more prolonged, louder, higher in pitch than the inspiratory. Abnormally, bronchial breathing signifies consolidation. This condition obtains in pneumonia, phthisis, atelectasis, infarct. Where air-vesicles still remain, consolidation being incomplete, the respiration is termed **broncho-vesicular**, or, by some, **rude, rough, or harsh**. Such a condition obtains in incipient phthisis.

Over a cavity with collapsing walls and free egress of air, a sound is heard resembling bronchial breathing and by many not differentiated from it, called by Flint **cavernous respiration.** The pitch, however, both inspiratory and expiratory, is lower than that of bronchial breathing, and the tubular character is less prominent.

By blowing over the mouth of, or into, an empty, narrow-necked bottle, a sound is produced, from the similarity to which, respiration heard over a large air-containing cavity with rigid walls, is called **amphoric.** There is to this sound a musical or metallic quality and echo peculiarly characteristic. This is best heard in pneumothorax.

When the inspiratory sound ceases before the inspiratory act is finished, and when the pitch is high and the quality tubular, the bronchial breathing denotes consolidation. If, on the contrary, the beginning of the inspiratory act is unheard, the pitch low, and the quality vesicular, dilated vesicles of emphysema are indicated. In noting the length of the expiratory sound, the pitch and quality are to be observed, a prolonged expiratory sound of high pitch and tubular quality being, usually, significant of consolidation, while of low pitch and vesicular quality it generally means emphysema. The fact that at the right apex there is, normally, a more prolonged and higher-pitched expiratory sound than at the left must be kept in mind.

Interrupted, jerky, or **cogged-wheel respiration** may be found in the nervous, those suffering the pains of acute pleurisy, intercostal neuralgia, etc. It is of little diagnostic value, save as an occasional confirmatory evidence of phthisis.

Adventitious respiratory sounds or rales are sounds without analogous normal sounds. They are new. The division into groups is here made on an anatomical basis.

The dry, wheezing, croupy respiratory sound of laryngeal obstruction and the moist rattle of fluid in the trachea, as in the death agony, furnish types of the dry and moist bronchial râles.

The moist bronchial rale is produced by the bubbling of air through thin liquid in the bronchus, this liquid being mucus, serous, bloody, or purulent in character, as in bronchitis, especially the chronic and purulent forms and in those forms attending phthisis, in œdema of the lungs, in some cases of pulmonary hemorrhage. In the larger tubes the bubbles are coarse, in the smaller ones fine, almost crackling in character, and usually spoken of as subcrepitant. Capillary bronchitis furnishes the most typical subcrepitant râle. Localized moist râles are always suspicious signs of tuberculosis. Fine râles, posteriorly, after a patient has been long in the recumbent posture, have no pathological meaning. The pitch of râles surrounded by consolidated lung is high. Coughing will often displace the fluid from the larger tubes and cause disappearance of the coarser râles.

Large, dry rales are snoring or almost booming in character, the smaller ones whistling, sibilant, or even squeaking. Constriction of the larger and smaller bronchi, respectively, produces these sounds. Acute bronchitis and asthma furnish, usually, both these varieties, as may constriction of a bronchus from external pressure.

The **crepitant rale** is the vesicular râle produced by the separation of the walls of the alveolus, made sticky by the commencing exudate of pneumonia. This separation occurs as air enters the alveolus at the end of inspiration. The râle is heard, therefore, at the end of inspiration, is fine, crackling in character. The sound can be imitated by rubbing the hair above the ear, between the thumb and finger. This râle is heard in the early stage of pneumonia, rarely in any other condition. It is to be carefully separated from the subcrepitant râle, which is moist, heard often, not alone at the end of inspiration, but as well at the beginning of, or during, expiration.

Cavernous rales are the gurgling sounds or bubbling produced in large cavities containing liquid, as in phthisis or bronchiectasis.

Pleural sounds are produced where there is a roughening of the pleural surfaces, as by the exudate in pleuritis, where there is heard a rubbing, coarse or fine, known as the pleural *friction* sound, or rub.

A metallic tinkling sound is at times produced where a large cavity containing air and, usually, liquid, as in pneumothorax, or a large phthisical cavity, communicates with a bronchus. Bubbles of air, passing through a little liquid in the bronchus, break through into the cavity with the above tinkling sound, or the fluid drops into the fluid in the bottom of the cavity with a metallic, splashing sound.

If a patient whose pleural cavity contains air and liquid, be shaken, a splashing sound is audible, known as the succussion sound.

Occasionally, sounds are met with, crumpling or crackling in character, whose anatomical location cannot be made out.

These are the indeterminate râles, at times pleural, at times vesicular, and, often, probably, in the smaller bronchioles. In the early stage of phthisis they may confirm other signs.

Voice sounds are diminished or suppressed by partial or complete obstruction of a bronchus, or by the interposition of a layer of air, fluid or solid, between the lung and the thoracic wall. Increase of transmission of voice sounds is, usually, due to consolidation, and when marked is termed **bronchophony.** If whispered words are distinctly transmitted to the ear the phenomenon is spoken of as **pectoriloquy.** **Ægophony,** heard at times just above the level of a pleuritic effusion, is a distant sound of a bleating character. Over large cavities with bronchial communication amphoric sounds are heard.

BRONCHITIS.

The features common to all forms of bronchitis are **cough** and **bronchial rales.**

The **cough** of acute bronchitis is at first tight, dry, and attended with a cutting, rasping pain, chiefly substernal. When the cough "loosens" the pain is less, and there is a frothy mucous expectorate that may become muco-purulent. In the chronic form there is usually, especially in the morning, an expectorate of a mucous, muco-purulent or sero-purulent character. Rarely a dry form with no sputa is met with, and, on the other hand, a form in which the cough is attended by the raising of a large amount of thin, sero-purulent fluid, giving the name bronchorrhœa to the affection.

In the **fibrinous bronchitis** fibrinous casts of the bronchi are raised. A fetid odor from the presence of putrefactive bacteria has caused one form to be spoken of as **fetid bronchitis.**

In all forms of bronchitis *râles* are heard, and these are **bilateral.** In the beginning of the acute inflammation the râles are sibilant or sonorous. Later the moist râles appear. It is quite characteristic of bronchial râles that coughing may cause their temporary disappearance. Chronic bronchitis will show few or numerous râles, depending upon the amount of liquid in the tubes and its degree of thinness. Thick mucus or the fibrinous plugs of plastic bronchitis will not cause moist râles.

It is important to remember that the râles of bronchitis are not associated with dulness on percussion, with increased fremitus, or with tubular breathing. The absence of these signs serves to exclude, in the case of the acute form, pneumonia, and in the chronic, tubercular consolidation. A localized bronchitis, especially when the pitch of the râles is elevated, always points suspiciously toward commencing tubercular consolidation. Examination for tubercle bacilli should always be made in such a case.

In **capillary bronchitis** the râles are fine, crackling, heard with inspiration and expiration—are subcrepitant. As this form is associated with pneumonic consolidation (lobular pneumonia), it will be spoken of under that head.

Acute bronchitis is a painful affection. The pain is described as burning, aching, located chiefly beneath the sternum and aggravated by coughing. At such times the "sore place feels raw." There is a sense of constriction in the chest, and often general malaise. Fever may be as high as 100° to 103°. There is often an accompanying or preceding rhinitis, pharyngitis, or laryngitis. In a few days the dry, irritating cough is supplanted by a moister, loose cough, the patient raises a little mucus and muco-pus, and obtains a sense of relief, from the cough.

The examination of the chest may show in children, increase in respiration rate, this being very marked if the smaller bronchioles are involed. Dry râles are at first heard, and later small and large moist ones. No appreciable change is noted in the percussion note unless capillary bronchitis and consequent consolidation are present. The rhonchial fremitus can be felt where the moist râles are large.

The chronic form of bronchitis is met with in those who have suffered from repeated attacks of the acute form. Cough is the prominent symptom. The health may be in other respects good. Being often coupled with more or less emphysema, or asthma, or cardiac difficulty, especially in the old, shortness of breath on exertion is often complained of. In some patients there is little complaint during the summer, but in the winter they have the "winter cough." The secretion from the bronchial mucous membrane may be very scanty—dry bronchitis—very profuse and serous in character—bronchorrhœa—fetid from putrefactive changes—fetid bronchitis. Usually it is muco purulent or purulent in character.

The physical examination reveals nothing save the râles, dry and moist, universal over the chest. Often, however, an alteration in the signs is caused by the accompanying emphysema, or heart disease, or bronchiectasis.

The only way in which an acute *fibrinous bronchitis* is to be distinguished from a severe attack of the acute catarrhal variety is by the expulsion of fibrinous casts of the smaller bronchi. The disease may be fatal. It is regarded as distinct from the fibrinous form where diphtheritic membrane develops on the bronchial mucous membrane by extension downward from the larynx and trachea.

Chronic forms of fibrinous bronchitis are occasionally met with where there are recurrent attacks resembling acute bronchitis, during which there are paroxysms of cough, bringing up sputa, mucus, blood-stained and containing casts of the bronchioles often rolled up into little balls. Occasionally the failure to get respiratory sounds clearly over a certain area of the chest may lead to the suspicion that the trouble is located in that particular region.

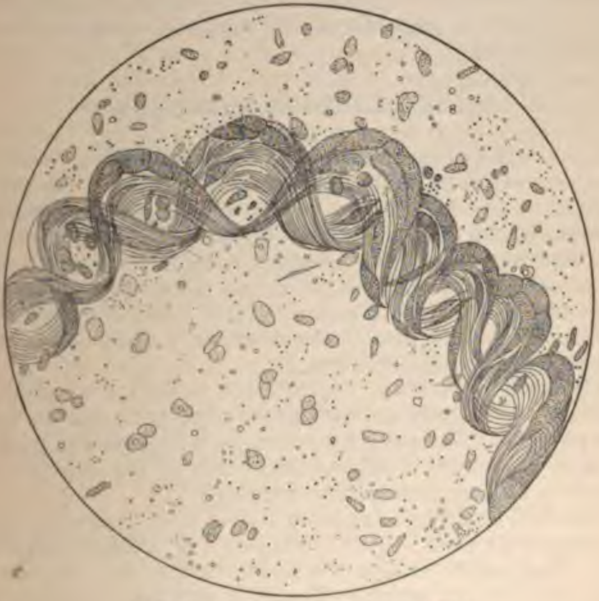
ASTHMA.

Asthma is a disease which, whatever its true pathology may be, presents little difficulty in diagnosis if a patient is seen during a paroxysm. The diagnosis can often be made by the sight of the patient and by the sounds heard even at some distance from him. The attack often comes on or is aggravated during the night. There is an intense hunger for air, a sense of impending suffocation. The sufferer sits up in bed, or sits at the open window, breathing laboriously, calling into play the extraordinary muscles of respiration. He is often covered with perspiration, the bloodvessels distended, and altogether he presents a most alarming appearance, and one well fitted to frighten the uninitiated into thinking death from suffocation is impending. There is a most marked expiratory dyspnœa. Wheezing and blowing sounds are plainly audible. Closer examination of the chest will show that in spite of the violent respiratory efforts there is comparatively little movement; the chest tends to remain in a condition of inspiration. If emphysema be likewise present the characteristic features of this disease are noted. There is no dulness upon percussion, but often an increased resonance. Rhonchial fremitus may be present, but the characteristic physical sign is the abundance of sibilant and sonorous râles heard throughout the chest, changing from time to time both their location and their character.

At times a probable diagnosis has to be made from the history of previous attacks, which patients are usually able vividly to describe. It is to be remembered that in the interval between two attacks a patient may present no sign of the disease. Not infrequently bronchitis and emphysema or cardiac disease are associated with asthma. The evidences of these diseases may be found not alone during the asthmatic paroxysm, but as well in the interval.

While of little or no value diagnostically, it is of interest to note that the sputa of asthmatics contain fibrinous threads wound spirally about a central thread, known as Curschmann's spirals.

FIG. 27.



Curschmann's Spirals. (VON NOORDEN.)

Charcot's crystals are also found, as well as eosinophilous cells. The blood of asthmatics, during the attack, may contain a large number of eosinophiles.

BRONCHIECTASIS.

The chronic bronchitis or interstitial pneumonia, with which bronchiectatic cavities are associated, may cause symptoms that overshadow those of the bronchiectasis. The cough which these patients have in the morning may bring up a large amount of pus or muco-pus, often of a putrid odor.

The signs are those of a cavity; if empty, with a tympanitic resonance and cavernous respiration; if full of fluid, dull, with lessened fremitus, and perhaps gurgling râles. The physical examination is often unsatisfactory in its demonstration of bronchiectatic cavities, and dependence for diagnosis must rest mainly upon the evidences of chronic bronchitis or interstitial pneumonia, and the history of expectoration, especially in the morning, when the cavities have had time to fill over night, of a large amount of purulent material, often of offensive odor.

ACUTE CROUPOUS PNEUMONIA.

In its typical form, as frankly expressed in vigorous adults, croupous or lobar pneumonia is one of the most readily recognizable of diseases. In young children, in the aged, in drunkards and those debilitated by other causes, it often pursues an irregular masked course, making its diagnosis a matter of considerable difficulty.

In the typical case there are seven classical symptoms—sudden onset, chill, pain in the chest, fever, cough, expectoration of rusty sputa, rapid breathing.

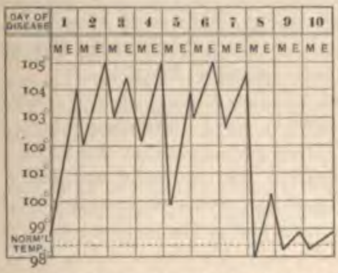
The **onset** is sudden. An exposure to cold, a wetting and chilling of the body are assignable exciting causes in not a few instances. Premonitory anorexia, headache, and general malaise are noted in some cases. But in the majority the attack is without premonitory warning.

The **chill** is severe, resembling that of malaria or of pyæmia. It is more than the "chilly sensations" of typhoid, tonsillitis, etc. The patient shakes, the teeth chatter, the lips become blue, and the skin is cold. The chill may last for an hour.

Pain may be noticed even during the chill. It is commonly referred to a point just below the right nipple, as the lower right lobe is the lobe most frequently inflamed. The accompanying pleurisy is regarded as the main cause of the acute pain. The site of the pain will vary, of course, with the lung and lobe involved. Diaphragmatic pleurisy may cause the patient to locate the pain in the abdomen. This readily leads to error in diagnosis unless the other symptoms be carefully weighed and the chest closely examined. In central pneumonia pain may be wanting, as it is in many of the atypical cases to be considered later. It is often the symptom of greatest importance from the patient's point of view, because of its intensity and persistence.

Fever is perceptible to the touch by the time the chill is ended. The temperature will have risen inside one hour, four to seven degrees—103° to 105°. It seldom falls, save as influenced by antipyretic measures, at any time during the illness, below 102°, and is often, especially at night, 104° or 105°. The termination of the fever is usually as abrupt as its first appearance. At about the end of a week there is a sudden defervescence with amelioration of other symptoms, the pulse becoming slower, breathing easier, and the patient feeling much more comfortable. Profuse sweating may occur at this time. This crisis is occasionally attended by evidences of collapse, subnormal temperature, feeble pulse, cyanosis. The crisis may occur even as early as the third day, or may be deferred until the tenth or twelfth. Termination by gradual defervescence—by lysis—is not so common as by crisis. Persistence of fever after two weeks or persistent rise of temperature after crisis should always excite suspicion of complications or unpleasant sequelæ, as abscess, delayed resolution, tuberculosis, empyema.

FIG. 28.



Pneumonia with pseudo-crisis on the fifth day.

Cough is often partially suppressed on account of the pain it causes. Within forty-eight hours the dry cough is succeeded by a moist cough, the sputa being viscid, tenacious, and colored by the sanguinary pigments a rusty brown color. This **rusty tenacious sputum** is almost pathognomonic of pneumonia. In the debilitated it may be thinner and of a prune-juice color. Children and the aged may fail to eject the viscid mass from the bronchi, or they swallow it instead of expectorating it. The diplococcus of Fraenkel can be found in the sputum.

Rapid respiration is an early symptom, persisting throughout the course of the disease. The rate may be but slightly increased, as to thirty, or may be as high as sixty to eighty, per minute. Usually it is about forty. In children, of course, it is more rapid. The breathing is quick and panting.

Among other features of the disease may be mentioned the increase in **pulse** rate. This is usually over one hundred, and may even in favorable cases remain at one hundred and twenty to one hundred and thirty. It is usually, in sthenic patients, full, strong, and bounding.

Nervous symptoms may be marked even from the first. Delirium, while it is commoner in the pneumonia of drunkards, is at times pronounced and violent in the so-called typical cases. Severe headache with delirium may easily mislead one into regarding the case as meningitis unless the chest be examined.

The **tongue** is coated, or, in severe cases with mouth breathing, dry and cracked. Vomiting is not rare at the beginning and during the course of the disease in drunkards and the over-medicated. Constipation is the rule.

Labial, nasal, and even genital **herpes** is very common and of diagnostic value. The skin is usually hot and dry, except at the crisis or during failure of the heart. The cheeks, especially the one on the affected side, are often flushed, the color at times being purplish in tint.

There is, in most cases, albuminuria. The **urine** is acid, of high specific gravity, urea increased, the chlorides diminished. Free diuresis is common at the time of crisis.

Blood examination, while rarely necessary as a confirmatory means of diagnosis, shows little change in the number or character of the red corpuscles, but a quite marked leucocytosis, at least, it is said by von Jaksch and others, in cases terminating favorably. The blood is very rich in fibrin also.

Physical Examination. Inspection shows the flushed cheek, rapid, panting respiration, often catchy from the effort to restrain the movements of the affected side, which are painful from the accompanying pleurisy. When consolidation is complete and extensive there may be restricted movement of the affected side, mensuration confirming the results of inspection.

Palpation. Vocal fremitus is perceptibly increased over the affected lobe. It is perhaps needless to say that vocal fremitus is most perfectly marked in the second stage—consolidation. In the first stage it is usually not increased, and as the lung clears up the exaggerated thrill gives way to the normal fremitus. In the later stage rhonchial fremitus can occasionally be made out where the moist râles are plentiful. Pleural friction fremitus is sometimes appreciable by palpation.

Percussion. In the early stage there is no change in the resonant note, or perhaps a slight elevation in pitch. Dulness soon appears, rapidly succeeded by flatness over the affected lobe. Immediately above the line of the pneumonic consolidation there is sometimes heard an hyperresonant tone, as also over the vicariously emphysematous sound lung. During resolution resonance gradually reappears in the affected lobe.

Auscultation. In the first stage the fine crackling sound heard at the end of inspiration and almost pathognomonic of of pneumonia—the crepitant râle—is heard. In the stage of red hepatization the vesicular murmur is entirely lost, the tubular, high-pitched bronchial breathing, with its prolonged expiration, being plainly heard and seeming to come from a point very near the listening ear. Voice sounds seem strikingly near, and are of a nasal character. Bronchophony is better marked in this disease than in any other. As resolution begins moist râles appear, subcrepitant in character. The râle redux—the returning crepitant râle—may also be heard. The moist sounds are at times quite coarse. The bronchial element gradually disappears, and vesicular sounds are again manifest. A friction rub can often be made out. It should be remembered by the student that the crepitant râle so often spoken of as the distinctive sign of pneumonia is only heard at the *beginning* of pneumonia, and more rarely at the end. Often, by the time the physician sees the patient, only bronchial respiration can be made out. And another point worthy of attention is that all parts of a lobe are not always in the same stage of inflammation. There may at times be heard in one and the same lobe the crepitant râle, bronchial breathing, the subcrepitant râle of beginning resolution.

In speaking of the physical signs it has been assumed that the pneumonia is not central, and that the bronchus is patent. **Central pneumonia**, where inflammation begins in the centre of a lobe, and where the diseased tissue is sur-

plugged, the vocal fremitus may be suppresse
voice sounds and the bronchial breathing.
dislodge the plug of mucus or exudate, and
phenomana again suddenly appear.

Clinical Varieties.

1. **Central Pneumonia.** This has already
as the form where the consolidation is first pres
of a lobe. The diagnosis in these cases must
upon the clinical history of chill, fever, coug
From non-involvement of the pleura, pain will
or absent. As the disease advances toward t
the lobe, physical signs become more evident.

2. **Drunkard's Pneumonia.** Drunkard's p
term applied to the disease as it appears in
chronic alcoholism, whether at the time of th
tient has been heavily drinking, or not. The in
are sometimes indefinite, the chill but slight,
very high. Delirium comes on early and ser
pain. The sputa are, in these cases, often thin
red or prune-juice color, or they may be sm
Gastric irritation is common. The pulse is ra
there is subsultus tendinum, great restlessness, i
delirium, giving the clinical picture of deliriun
less the chest be carefully examined, the pneu
readily overlooked, attention being attracted
potu. In all cases of delirium tremens the ph
be on the lookout for pneumonia. The progno
of pneumonia is grave.

may take the place of the chill. The fever is high, the pulse full and bounding, respiration from fifty to eighty, and expiration often attended by a little moan. The child may be delirious. The sputa are swallowed, but there is an annoying cough, and tough, viscid mucus can be seen in the throat during the act of coughing. Vomiting, or diarrhœa may be excited by the mucus swallowed. The upper lobe is oftener affected in children than in adults. There is rarely the same satisfactory result obtained from a physical examination as in the case of an adult. The child may struggle and cry, though some regard this struggling and crying as an evidence against pulmonary disease; and the puerile respiration and naturally great resonance of the chest serve to obscure the slight dulness and tubular breathing that may be present. Brain symptoms may be so prominent as to simulate meningitis.

5. **Typhoid Pneumonia.** Typhoid pneumonia is a term applied to the asthenic or adynamic type of the disease. It is to be carefully distinguished from the cases of pneumonia and typhoid fever, where the two diseases are present at the same time.

Complications and Sequelæ.

Numerous organs may be involved in the course of an acute pneumonia. The pleurisy, usually fibrinous, may become serous or suppurative. In the pus of the latter cases the diplococcus is generally found. Pericarditis is common, as is endocarditis, the latter often of the malignant type. Meningitis is a fatal complication. Jaundice is common in some epidemics, its exact pathology in cases of pneumonia being still *sub judice*. Renal complications are rare. Relapse is rare, recurrence common.

Terminating in resolution, there is often a remarkably quick clearing up of the lung, the physical signs rapidly becoming normal. By **delayed resolution** is meant the persistence of slight dulness, moist râles, broncho-vesicular respiration, with little or no fever, and final recovery. Localized, or diffuse infiltrating, suppuration is occasionally met with as a sequel. The coughing up of a large amount of pus, containing elastic fibres and the signs of localized fluid before, and of a cavity after, the expectoration, may enable the physician to make a diagnosis of the case, which he may have suspected from the continuance of the fever, the sweats, irregular chills, and the great prostration. Gangrene can be recognized by the odor of the breath and the presence in the expectorate of blackened lung tissue.

Interstital pneumonia, or fibroid induration, may follow an acute lobar pneumonia, and tuberculosis may become implanted in a lung weakened by pneumonia, or a latent tuberculosis may be lighted up afresh by the acute disease.

The pathological condition, the physical signs attending it, and the symptoms commonly present may be grouped as follows:

Pathological conditions.	Physical signs.	Symptoms.
1. Stage of congestion, exudation beginning, but not filling air vesicle.	Crepitant râles, slight dulness, at times pleural friction.	Chill, pain in side, sudden rise in temperature, hacking cough, slight dyspnœa.
2. Stage of red hepatization; lobe consolidated, air vesicles filled with exudate.	Vocal fremitus increased; dulness on percussion; bronchial respiration and bronchophony; at times pleural friction.	Cough, with rusty sputum, rapid respiration, with dyspnœa; fever continuous, 102°-105°.
3. Stage of gray hepatization or of resolution; air again enters the vesicle.	Same as in second stage, though gradual or rapid approach toward normal, *i. e.*, fremitus and dulness, lessened; broncho-vesicular respiration, subcrepitant râles.	Sputa become less rusty, more yellowish; fever high, but dropping by crisis (or lysis) to nearly normal, with slowing of pulse and respiration-rate.

Differential Diagnosis. Pneumonia is less often mistaken for some other disease than some other disease is regarded as pneumonia. Usually very readily diagnosticated, it is easily overlooked in drunkards, in children, in the aged, in those suffering from some debilitating disease, as diabetes or typhoid fever, where it occurs associated with tuberculosis, and where it is central or where the main lobar bronchus is occluded.

Among the diseases that may be mistaken for pneumonia are the following:

Œdema of the lungs, that may come on suddenly, as in the course of a nephritis or cardiac disease, and is attended with dyspnœa, cough, expectoration. But in this condition fever is absent, cyanosis is marked, the sputa are frothy and not rusty-colored, the râles are subcrepitant, or coarse moist râles, and are heard on both sides of the chest. The dulness and bronchial respiration of pneumonia are lacking.

Pulmonary Engorgement; Hypostatic Congestion. In protracted fevers, in cardiac disease where bed-ridden patients remain for a long time upon their backs, there can be heard posteriorly, especially on deep inspiration, a crackling sound indistinguishable from the genuine crepitant râle of pneumonia. Slight dulness and even a harsh respiratory sound can be made out at times. This is due to the feeble cardiac action and consequent engorgement of the bloodvessels in the dependent portions of the lung. It is bilateral, unattended by rusty sputum or by additional rise in temperature.

Acute Bronchitis. In children especially, this can easily be looked upon as pneumonia. There is absence of severe chill, the pain is substernal at first, the râles are bilateral, there is no dulness or bronchial respiration. Respiration is not usually as rapid, nor fever so high.

Pulmonary Apoplexy. This accident, usually in connection with cardiac disease or peripheral thrombosis, produces sudden pain, dyspnœa, cough, and expectoration, and the physical signs somewhat closely resemble those of pneumonia. But there is less fever, dyspnœa is intense at first and gradually becomes less; the sputa, instead of being viscid and containing rusty-colored pigment, are thinner and contain pure blood. It must not be forgotten, however, that pneumonia may develop at the site of a pulmonary hemorrhagic infarction.

Typhoid Fever and Meningitis. Typhoid fever and meningitis are closely simulated by some cases of pneumonia. The examination of the chest should establish the diagnosis of pneumonia and the absence of distinctive features of typhoid, as slow onset, rose-spots, or of meningitis, as retraction of the neck, strabismus, paralysis, etc., serve to exclude these diseases. The question of whether the two diseases co-exist must at times be held in abeyance until the case has been carefully watched for several days.

In cases where resolution does not occur, examination for tubercle bacilli should be repeatedly made, as phthisis may develop rapidly with initial chill, fever, and evidences of consolidation, and be at first indistinguishable from croupous pneumonia.

For broncho-pneumonia and pleurisy *vide* pp. 176 and 193.

BRONCHO-PNEUMONIA; CAPILLARY BRONCHITIS.

Inflammation of the capillary bronchioles is so commonly, if not invariably, associated with or followed by, alveolar hepatization, and the clinical features of the disease are so impossible of distinction from those of the accompanying pneumonia, that the two affections are best regarded as one and the same, and the term broncho-pneumonia, at once implying the origin of the pneumonic process and the association of the bronchi and alveoli in the inflammatory action, is to be preferred to capillary bronchitis or to lobular or catarrhal pneumonia, names often applied to this affection.

It is met with oftenest in children and the aged, following an ordinary bronchitis, especially that accompanying measles, whooping-cough, influenza, and diphtheria. Phthisis may begin as a tuberculous broncho-pneumonia. Inhalation pneumonia is in reality a broncho-pneumonia.

The disease is preceded by signs of an ordinary bronchitis or "cold on the chest." Frequently there are the cough and other evidences of the bronchitis of measles or pertussis. The onset of capillary bronchitis is marked by a rise in temperature rarely preceded by a chill, increased pulse-rate, and, above all, by rapid and dyspnœic breathing. Examination now shows, in addition to the signs of the primary bronchitis, the fine subcrepitant and, perhaps, sibilant râles, indicating the invasion of the smaller tubes. These signs are best marked posteriorly, and are bilateral. Later, there may be made out, if the alveoli involved be superficial and the group of sufficient size, the signs of consolidation—dulness, harsh respiration, increased fremitus and voice sounds. Often, however, the hepatized areas are so small that no changes in physical signs, appreciable by the ear, are produced. Dyspnœa, in the severer cases, is marked, and may be extreme. There is constant, distressing cough, continuance of fever, rapid pulse; cyanosis may be marked, and with this appear greater prostration, restlessness, and anxiety. Death is preceded by right heart dilatation. And larger râles are now heard, due to the accumulation of mucus in the larger bronchi, the weak tussive efforts being now insufficient completely to evacuate the bronchi.

Broncho-pneumonia may easily be confounded with croupous pneumonia or with bronchitis. From lobar pneumonia it is distinguished as follows:

Lobar Pneumonia.	*Broncho-pneumonia.*
1. Onset sudden, with chill.	1. Onset gradual, with bronchitis.
2. Pain usually marked.	2. Pain not marked.
3. Rusty sputum, with diplococcus.	3. Sputa mucous or muco-purulent, often with streptococcus.
4. Termination by crisis.	4. May last many weeks, defervescence gradual.
5. Usually unilateral.	5. Bilateral.
6. Dulness, bronchophony, bronchial respiration over a lobe, and marked	6. Dulness, bronchophony, bronchial respiration over small areas; sounds often faintly perceptible from small area involved and from accompanying vesicular sounds.
7. Crepitant râle at beginning.	7. Subcrepitant râle at beginning.

From bronchitis affecting the larger tubes, it is distinguished by the higher fever, more rapid respiration, greater dyspnœa, fine character of the râles, and the presence, in many cases, of evidences of consolidation.

CHRONIC INTERSTITIAL PNEUMONIA.

Chronic interstitial inflammation of the lung—cirrhosis—may follow pneumonia, either the croupous variety or bronchopneumonia; it is not infrequently the result of the inhalation and lodgment in the lung of foreign material, such as coal-dust, bits of stone or metal, the production there of a reactive inflammation and overgrowth of new connective tissue (pneumokoniosis); it may arise from a primary pleurisy, and not infrequently primary pulmonary tuberculosis is associated with fibroid induration (fibroid phthisis), producing the same mechanical effects in the chest as are produced by the ordinary cirrhosis. Whether this trouble is primarily non-tuberculous or not, it is true that in most cases tuberculosis is found in the later stages of the disease. It is usually unilateral, slow in its progress, and accompanied by contraction of the connective tissue, with resulting deformity of the chest walls and deformity and displacement of the intra-thoracic organs. In this way, the pericardium, with the heart, may be drawn toward the affected side; bronchi may be dilated by the inflammatory changes in the peribronchial tissue, and bronchiectatic cavities may result.

Inspection in these cases will reveal a sinking in of the affected side and its comparative immobility. The apex-beat may be displaced toward the affected area and spinal curvature may have resulted from the contracting connective tissue drawing the column over. The convexity is toward the sound side.

Palpation confirms what has been detected upon inspection. Fremitus is increased over the diseased lung. A rhoncial thrill is often discerned by the palpating hand.

Percussion yields dulness. While this is the rule, there may be a resonant, high-pitched, tympanitic tone, due to the air in vesicles that are not yet destroyed, and to that in the dilated bronchi. This is often very deceptive, as advanced disease may exist and no dulness be perceptible.

Auscultation shows a loss of the pure vesicular tone. There is a bronchial element to the sound, and where pulmonary cavities or bronchial dilatations are large the murmur may partake of the amphoric character. The moist râles are often abundant, and dry râles are frequently heard.

The disease is not incompatible with active labor for years. There is cough with expectoration, more or less profuse, the quantity depending largely upon the severity of the coexisting bronchitis and the degree to which bronchiectasis exists. Exacerbations are common. There may then be fever, profuse expectoration, night sweats, rapid loss of strength. Rallying from these attacks, the patient again goes about his work, though month by month there is a little falling off in weight and strength, and dyspnœa becomes more marked.

Diagnosis in advanced cases is easy, from the result of physical examination. Tubercle bacilli should be sought for, and in the true fibroid phthisis they are usually found, and late in the forms of cirrhosis following pneumonia, pleurisy, or anthracosis they can be detected.

PULMONARY EMPHYSEMA.

The physical signs of this disease are clearly understood if the pathological condition present is kept in mind. The air vesicles are distended, their walls lack elasticity, and the entire lung occupies a greater space than normal, the lung shelving over the cardiac region and reaching at times an inch lower than normal.

Inspection. The countenance of the patient is dusky, the lips full, the neck often short, the sterno-mastoid and even the scaleni muscles standing out prominently. The shoulders are stooped and the chest has a rounded, barrel-shaped look from the increased antero-posterior diameter. In its movements it lacks the expansive motion of the healthy chest, moving up and down as a solid bony framework. The lower part is often seen to be retracted instead of expanded during inspiration. The epigastric angle is more obtuse than normal. The veinlets along the line of the costal arch are often dilated; the apex-beat is seldom seen, though epigastric pulsation and pulsating veins in the neck may be visible when there is right heart dilatation. Expiration is seen to be much prolonged.

Palpation may fail to locate the apex-beat, though epigastric pulsation may be distinctly felt, and, as often happens in the late stages of the disease, an enlarged liver, from passive engorgement can be made out. The vocal fremitus is usually diminished.

Percussion reveals resonance such as is heard over air-containing cavities that are not vesicular. The vesicular element is not, however, entirely absent, so that very properly "vesiculo-tympanitic" is the term employed to designate the resonance of emphysema. It is very characteristic, also, to find resonance over a wider area than the normal. Cardiac dulness may be hidden beneath resonance; anteriorly and posteriorly the pulmonary resonance may extend a full inch lower than usual; hepatic flatness may begin at the seventh or even eighth rib in the right mammillary line. The liver may be appreciably displaced by the pressure of the distended lungs, and especially when it is passively congested from the dilated right heart it forms a mass below the costal arch plainly perceptible to touch and sight, and whose dulness is clearly marked.

Auscultation. So feeble is the respiratory sound in emphysema that it is sometimes scarcely audible. The expiratory sound is markedly prolonged, as it is in bronchial respiration, but, unlike that variety, it is soft and of low pitch. Fine crackling sounds are sometimes heard, resembling fine pleuritic sounds or crepitant râles. Their mode of production is unknown. Emphysema is often the terminal event in cases of chronic bronchitis and asthma. Not infrequently, therefore, the moist or dry râles of these two diseases are heard in cases of emphysema. And in all cases of chronic bronchitis or of asthma, where there is marked dyspnœa, cyanosis, pulsating cervical vessels, careful examination of the naked chest should be made for evidence of emphysema. In case cardiac dilatation has become excessive a tricuspid regurgitant murmur may be made out. The great obstruction to pulmonary circulation shows in the accentuation of the pulmonic second sound.

The diagnosis is made upon the results of physical exploration. The history in these cases is usually of shortness of breath on exertion, cough, palpitation. Recurrent attacks of bronchitis and of asthma are common. When the right heart becomes dilated the symptoms become aggravated, and are those of venous obstruction. The portal system is congested, producing enlargement of the liver, gastric and intestinal derangement, and often dropsical symptoms. Death usually occurs as in cases of uncompensated valvular disease.

Senile or **Atrophic** emphysema is sometimes met with in the old, presenting about the same symptoms and signs as the hypertrophic variety save in this important particular, that the lung space is not visibly increased, so that there is a lack of the barrel-shaped chest and the increased area of pulmonary resonance.

PULMONARY HEMORRHAGE.

1. Pulmonary Infarction—Pulmonary Apoplexy.

From the peripheral veins the site of a thrombosis, or from the right heart containing a thrombus, as it may in valvular disease of the heart and especially in mitral stenosis, an embolic plug blocking up a pulmonary artery may arise and cause hemorrhagic infarction. Small emboli may cause no clinical symptoms. Larger ones, as in the main pulmonary artery, may produce sudden death. The accident can be suspected where a patient, with valvular disease, has a sudden dyspnœa, perhaps pain in the chest, and begins soon after to cough up bloody sputum, perhaps slightly mixed with mucus.

Large peripheral infarcts may be recognized by dulness on percussion and by moist râles, fine or coarse. Signs of pleurisy may be evident a few days later, as a plastic pleurisy often follows the infarction.

2. Hæmoptysis.

Where blood enters the bronchi and is coughed up, the hemorrhage is spoken of as hæmoptysis. A knowledge of the causes producing such hemorrhage is of aid in diagnosis, and conversely the hemorrhage may be of value in enabling us to establish a diagnosis of some pulmonary or thoracic disease. The commonest causes are (1) tuberculosis, either early or late; (2) other pulmonary diseases, as pneumonia, cancer, abscess, gangrene, infarctus; (3) heart diseases; (4) laryngeal, tracheal, or bronchial ulcerations; (5) aneurism; (6) vicarious hemorrhage, taking the place of the menstrual discharge; (7) hemorrhagic diseases. A pathological basis is undiscoverable in some cases.

There is seldom any difficulty in determining that the blood escapes from the mouth by way of the larynx. The patient is conscious of *coughing* up the blood. The blood tastes warm and sweet. It is found to be alkaline, usually bright red, and mixed with air, and perhaps muco-pus in case it comes from pulmonary cavities or from bronchi, the seat of a bronchitis. Blood may be lost in sufficient quantity to induce signs of acute anæmia that may be fatal in its consequences. Death, when it does occur, is oftener attended with signs of suffocation from filling of the bronchi and alveoli with blood. For some days after an initial hemorrhage dark clots of blood may be coughed up.

Hæmoptysis has to be distinguished from hemorrhage from the nose, naso-pharynx, mouth, and the stomach. Examination of the nose, mouth, and pharynx, employing the rhinoscope if necessary, will serve to exclude hemorrhage from these sources. Hemorrhage from the stomach is attended by vomiting of blood, often acid, dark and coagulated, and mixed with stomach contents. (*Vide* table, p. 106.)

ŒDEMA OF THE LUNGS.

Œdema of the lungs, while not a primary disease, is a condition so frequently met with as an accompaniment or consequence of other diseases, that its recognition is of great importance. Œdema of the lungs is the result of the transudation of serum from the pulmonary vessels into the alveoli, bronchi, and alveolar walls. During inflammations, as croupous pneumonia, broncho-pneumonia, tuberculosis, abscess, and with neoplasms and infarcts, there is more or less localized œdema in the neighborhood of the pathological lesion.

Congestion and œdema usually, if not always, go hand-in-hand. General œdema is seen where death occurs from gradual cardiac failure in cases of heart disease, valvular or myocardial, in many of the general diseases, or those attended by great anæmia and cachexia. It may suddenly develop in the course of a chronic nephritis.

Symptomatically there is dyspnœa. This may be slight or of extreme degree, inducing cyanosis and orthopnœa. There is cough, attended by the expectoration of a frothy, watery, perhaps bloody, fluid. The chest is filled with moist râles of every variety, in extreme cases the large bronchial or the tracheal râles drowning the sounds of all the other smaller ones. There may be such marked congestion or such an accumulation of fluid in the alveoli and bronchi as to cause some dulness in the dependent portions of the chest.

GANGRENE OF THE LUNG.

Pulmonary gangrene is met with as a sequel of croupous pneumonia in a small percentage of cases. It may occur after an inhalation pneumonia, or when foreign and putrefactive matter enters the lungs, as from perforation of the œsophagus or from an embolus. In the debiliated it occurs much oftener than in the previously strong, and it is to be regarded as due to the action of putrefactive germs upon a part whose resistance is lowered by depressed vitality.

The condition is recognized by the intensely fetid odor of the breath and by the sputum. The expectorate is offensive, often profuse, and contains elastic fibres, fatty crystals, granular matter, and pigment grains. The shreds of lung tissue can be seen oftentimes with the unaided eye as black, necrotic masses in the sputum.

The patient is prostrated, with evidences of profound septic intoxication. The temperature, usually elevated, may be subnormal, the pulse rapid and weak, and the patient gradually sinks. Delirium is occasionally seen. Evidences of a cavity may be made out if the necrotic area was superficial and of any considerable size. Moist râles can be heard in the neighborhood of this area, indicating the accompanying bronchitis.

ABSCESS OF THE LUNG.

Pulmonary abscess may follow croupous, broncho-, or inhalation pneumonia, may be embolic in origin, the original focus of suppuration being in some distant organ, or it may arise from traumatic puncture of the lung or rupture into it of an abscess in some neighboring organ, as the liver.

The fever, chills, sweats, characteristic of suppuration are usually present, though in some cases only slightly marked. The signs of localized fluid with surrounding consolidation (pneumonic or atelectatic) may in cases be made out, the exploring needle revealing pus. The coughing up of a large amount of pus with subsequent raising of pus, often fetid, and containing elastic fibres and bits of lung tissue, renders the diagnosis easy. Cavernous signs can be made out after evacuation of the abscess. Centrally located abscesses, or those of small size, cannot always be recognized until after the evacuation of the pus.

PULMONARY TUBERCULOSIS.

From a clinical standpoint, and in a measure from a pathological standpoint also, four forms of pulmonary tuberculosis are recognizable—(1) acute phthisis, (2) chronic phthisis, (3) fibroid phthisis, (4) miliary tuberculosis.

1. Acute Phthisis.

Acute phthisis is the ordinary galloping consumption. It is commonly frank in its expression, plainly showing the involvement of the lungs by cough, expectoration, and somewhat hurried breathing. The onset may be sharp, resembling that of pneumonia in its chill, rapid rise of temperature, and rapid respiration. In fact, a quite extensive tubercular pneumonia is often present at the beginning. Pain may or may not be noticeable. In other cases the symptoms are more those of typhoid fever, a premonitory headache, malaise, anorexia, gradual development of fever, and cough. In acute phthisis the strength fails quite rapidly. Emaciation is pronounced. The fever is continuous, usually higher in the afternoon, often reaching 103° to 105°, dropping toward morning to, or nearly to, normal, with perhaps profuse perspiration. The pulse is rapid; the cough is annoying, soon bringing up muco-purulent sputa in which, after breaking down of lung tissue, elastic fibres and tubercle bacilli are found. The disease may run its course in from three weeks to a few months. Occasionally a stormy onset is followed by symptoms of florid phthisis, which gradually subside, and the disease assumes the chronic form.

Tuberculous infection follows, not rarely, such diseases as measles and whooping-cough, where there is marked bronchial irritation and inflammation, inviting tubercular localization, or it may occur during the course of, or convalescence from, other acute infectious diseases, as typhoid fever.

The physical signs are those of consolidation, usually primarily at one apex. Large areas of tubercular pneumonia may resemble perfectly the consolidation of croupous pneumonia, the deception being rendered more complete by the sudden onset with chills, high fever, rapid breathing, blood-stained expectorate. In some of these cases only a careful watching, with repeated examinations of the sputa, renders a positive diagnosis possible. Death sometimes occurs even before bacilli and elastic fibres can be found. It is perhaps needless to repeat that the physical signs of such consolidation would be dulness, increased fremitus and voice sounds, tubular breathing.

In other cases physical examination may for some days be negative, or only a few moist râles, such as are found in non-tubercular bronchitis and broncho-pneumonia, may be discovered. Soon, however, consolidation is manifested by dulness, and harsh, prolonged, and high-pitched expiratory blowing. The disease makes rapid progress, cavities forming even inside of a few weeks.

From **croupous pneumonia** acute phthisis is to be distinguished by its oftener involving the apices, often only a part of one lobe, by the more remitting character of the fever, by the absence of the diplococcus, and presence, at least later, of the tubercle bacillus, by the absence of crisis, and the evidences of invasion of new lung tissue.

From **broncho-pneumonia** the progressive emaciation, hereditary history, tubercle bacilli, perhaps signs of a cavity, enable us to distinguish it.

From **typhoid fever** with extensive bronchitis it is distinguished by the signs of consolidation, by the tubercle bacilli, and the absence of the distinctive typhoid marks, roseola, tympany, etc.

2. Chronic Pulmonary Tuberculosis.

This disease, the ordinary "phthisis" or "consumption" of the laity, often presents to the physician symptoms so characteristic as to render a physical examination almost unnecessary save to determine the extent of damage done in the lungs. Consumptives often fail to recognize the serious nature of their ailment, because of the apparently trivial character of the initial symptoms and the slow and insidious progress of the disease. The subjective history in these cases, elicited by the careful inquiry of the examining physician, is in many instances quite convincing as to the existence of tuberculosis. There has been a cough, at first dry, hacking and irritating, and only troublesome on exertion or on inspiring cold or dusty air. Later the cough becomes more constant, sputa are raised especially in the mornings, and these have latterly been of a yellowish tinge. With the gradual failure of appetite there has been emaciation, attended by some loss of strength and power of endurance, and an increasing pallor of the skin. Dyspnœa and palpitation may be complained of. Toward night there has been a little fever; night sweats are common. Perhaps the coughing up of a little blood has alarmed the patient, or the statement of some friend, who has seen the patient after an absence of some weeks or months, that there is a great change in the looks, drives the previously unsuspecting, but now frightened, sufferer to the physician.

In other cases the subjective symptoms are slight, a trifling cough, a deranged appetite, a faint feverish flush toward night, the loss of a previously ruddy complexion—so slight in fact, so little positively indicative of tubercular disease of the lung, that the physician, unless he be wary, may neglect the examination of the chest, or upon hasty examination fail to detect the small area of disease. It is in these latter cases of incipient phthisis that early and accurate diagnosis is of the utmost importance, for upon it will depend the proper treatment of the case and the hope of a cure.

Heredity plays a not unimportant part in the history of many—25 per cent.—cases. It is astonishing how patients will hide the fact of the presence of consumption in the family. The most searching examination is often necessary in order to reveal the hereditary taint of tuberculosis in the form of consumption, cold on the lungs, hip-joint disease, white swelling, spinal curvature, scrofula, etc.

The **previous condition** and health of the patient may show some disease or environment favoring the development of phthisis. Previous catarrhal inflammation of the respiratory tract, depressing diseases, *e. g.*, diabetes, lowering the vitality and the resisting power of the organism, render the lungs peculiarly liable to localization of tubercle bacilli and favor their growth. In the same way certain occupations predispose to tuberculosis, by the constant irritation and trauma produced by the inhalation of foreign particles. Thus, miners, stonecutters, workers in glass, are often the victims of chronic pulmonary tuberculosis.

No **age** is exempt, though it is oftenest found in young adults.

Cough is usually slight at first, and may, indeed, though rarely, be comparatively insignificant during the course of the disease. It oftentimes disturbs the sleep at night, or is worse in the morning.

The **sputum** is at first slight, or even lacking; later it becomes mucous and glairy, and, when there is destruction of lung tissue and the formation of cavities, it is increased in amount and muco-purulent in character. The sputum, when there are cavities of any considerable size, is nummular, greenish-yellow in character, and contains heavy masses that sink to the bottom of the sputa cup. Blood is oftentimes coughed up in slight quantity, merely streaking the sputa. Coughed up in larger quantities, it constitutes the dreaded **hæmoptysis** of phthisis. Hæmoptysis may be the earliest symptom. The amount of blood coughed up may be very slight, or so large in amount as to induce syncope. Blood from the lungs is usually bright red, alkaline in reaction, mixed with air. Following the coughing up of blood the patient expectorates for several days small masses of clotted blood.

Microscopical Examination. The sputum of pulmonary tuberculosis contains two substances characteristic of the disease: elastic tissue and tubercle bacilli. For the detection of elastic tissue, one of the little masses from the sputum can be picked out with the forceps and then flattened between two glass slides or two glass plates. A low power detects, especially when the plates are held against a blackened surface, branching elastic alveolar tissue, or long, narrow, closely packed bronchial fibres. The detection of this elastic tissue is conclusive evidence of bronchial erosion or softening of the parenchyma of the lung. Red corpuscles, leucocytes and bacteria of various varieties are also found in the sputa of tubercular patients. The detection of the tubercle bacillus may be the only evidence of the existence of pulmonary tuberculosis, even before the clinical history or physical examination reveals the presence of the disease.

Method of Staining Tubercle Bacilli:

Three solutions are necessary.

Solution (*a*) "*Carbol-fuchsin.*

Saturated alcoholic solution of fuchsin	10
Aqueous solution of carbolic acid, 5 per cent.	90

Solution (*b*).

Strong hydrochloric acid	1
Alcohol, 95 per cent.	30
Distilled water	70

Solution (*c*).

Saturated aqueous solution of methyl-blue (filtered).

Solution (*a*) is best when freshly prepared.

1. A small quantity of sputum (the thicker yellowish part is the best) is taken with a forceps, placed on a thin cover-glass, and pressed into a thin film by a second cover-glass, laid over the first, and the two glasses then drawn slowly apart.

2. Fix by passing the cover, film side up, slowly through the flame of a Bunsen burner or alcohol lamp three times.

3. The glass is covered to its edges with several drops of staining fluid *a*, then held over a flame until it steams or boils slightly.

4. Wash by holding the cover under the hydrant, presenting the edge of the glass to the stream of water.

5. Decolorize by waving the cover back and forth in solu-

tion *b* (using an excess of the solution), until the red color has disappeared. By this procedure only the tubercle bacilli remain stained.

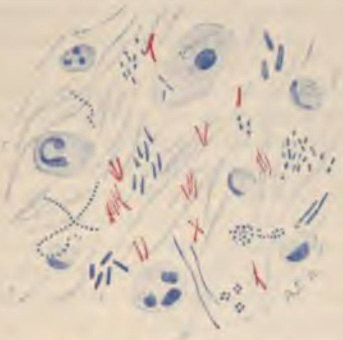
6. Apply solution *c* in the same manner as solution *a*, warming gently before washing off. Wash as in 4.

7. Dry in the air or by gentle heat.

8. Place a drop of Canada balsam upon the centre of the glass slide, invert the dry cover upon this drop and examine. Throughout all the process the cover should be held with forceps, and the film side should always be upward.

By this method of staining the tubercle bacilli are stained red by the fuchsin; other structures are stained blue by the methyl-blue solution, which is known as the counter-stain.

FIG. 29.



Tuberculous sputum stained by Gabbett's method (fuchsin and methyl-blue). Tubercle bacilli seen as red rods; all else is stained blue. (ABBOTT.)

Among the other symptoms of phthisis must be mentioned *fever*, which is rarely absent during the course of the disease. Frequently the thermometer is necessary in order to detect the slight evening rise of temperature; at other times the patient is conscious of a sense of heat and a flushing of the face, especially toward evening, only relieved by the copious sweat that occurs usually during the early hours of the morning. The evening temperature in these cases may be as high as 103° or 104°. Usually there is a morning drop in temperature to normal, or even subnormal; in other cases, especially where cavities are formed, where pus is abundant, and free absorption is taking place, there is a constant temperature of a remittent and not intermittent type. Along with the fever of phthisis there is progressive emaciation and weakness. The patient becomes pallid, and an examination of the blood will show a reduction in the number of red blood-corpsules, and in the percentage of hæmoglobin; in other words, there exists a secondary anæmia.

A *rapid pulse* is so commonly found in chronic pulmonary tuberculosis that it is really of diagnostic importance. In no condition, perhaps, apart from cardiac disease, can so rapid a pulse be found in a patient who is not ill enough to be confined to bed. Oftentimes the patient will walk into the office, making but slight complaint of illness, and yet the pulse will be found beating from 130 to 140 times per minute.

The *night sweats* of phthisis that have been spoken of may occur at any time during the course of the disease, though they are usually phenomena of the later months of the illness. The sweats are copious, the night clothes are drenched, and the patient is, or at least thinks he is, very much weakened by them.

Physical Examination. A well-advanced case of pulmonary tuberculosis will be taken as the type of the disease. Here inspection shows a depression and loss of motion at one apex, most marked in the infra-clavicular region. The loss of motion can sometimes be best observed by standing behind the patient and looking down upon the anterior chest. Over the affected area the vocal fremitus is increased, as detected by palpation. Palpation may also show a lessened expansion of the affected apex. Percussion reveals a high-pitched, dull note. Upon auscultation there is heard bronchial breathing with its prolonged and high-pitched expiratory murmur. Moist râles, chiefly of the subcrepitant variery, are detected, and at times sibilant and sonorous râles. Bronchophony is usually well marked. These signs are merely those of consolidation, and are not characteristic of tubercular consolidation. They are usually first marked at the apex of one lung, gradually extend over the entire lobe, and then are perceptible in the apex of the opposite lung. In advanced cases these signs are found over still wider areas. As cavities form, new signs are apparent. Over a cavity of sufficient size to be detected, there is tympanitic resonance upon percussion. The breath sounds are of the amphoric or cavernous variety. Where the cavity has yielding walls and is situated near the surface, the cracked-pot sound can sometimes be elicited. The râles in the advanced stage of the disease are frequently coarser than in the earlier stages. The recognition of consolidation in the typical case and in the advanced stages of the disease is usually a matter of little difficulty.

In the early stage, before the signs are well marked, the area of consolidation may be so slight or so centrally located as to defy the most careful examination. The apex of either lung should be carefully examined, especially by auscultation. Loss of motion, change of contour, alteration of tactile fremitus, may not be perceptible. Even percussion may fail to reveal any alteration in the note. Careful listening with the stethoscope will, however, usually show a harsher character to the respiratory sound, a slight prolongation and elevation of the pitch of the expiratory murmur. If these alterations in sound are associated with the presence of a few moist râles, especially the subcrepitant râles, the suspicion is strong of tubercular consolidation. In all doubtful cases the sputum should be examined for the presence of the bacillus tuberculosis. While the apices are the parts of the lungs oftenest primarily affected, it should never be forgotten that the apex of the lower lobes, or in fact any portion of the lung, may be first attacked. It should also be remembered that a dry pleurisy, especially if it affects one apex, is to be looked upon with suspicion as being probably of a tubercular character, and perhaps the starting-point of a pulmonary tuberculosis.

Attention should be called to the fact that normally the right apex exhibits a percussion note of a higher pitch and a respiratory sound of a slightly harsher character than does the left apex. Examination in doubtful cases should always be made carefully over the apex of the lung posteriorly, in the supra-spinous fossa, for it has been shown that the tubercular process oftenest commences nearer the posterior than the anterior surface of the lung (Taylor), so that it can be detected here when examination of the anterior wall of the chest may fail to reveal any abnormal sign.

It will be seen from what has already been stated that the diagnosis is usually readily made by the symptoms of cough, hæmoptysis, fever, emaciation, rapid pulse, which call our attention to a disease of the chest, and by the physical examination which shows the evidences of consolidation. In a few cases, however, the patient's attention is so directed to some organ other than an intra-thoracic organ, that the physician, unless he be on his guard, will fail to examine carefully the chest, and will overlook the disease, and it is remarkable how often patients strive, consciously or unconsciously, to call our attention away from the chest when they are somewhat fearful that they are the victims of beginning consumption. The stomach is frequently disordered. There may be complaint of a poor appetite, distress after eating, and even nausea and vomiting; in other words, the symptoms of a chronic or subacute gastritis. The bowels, too, are oftentimes disordered; there may be constipation or diarrhœa, the latter frequently due to a secondary tuberculosis of the intestines. This tubercular lesion of the intestines must always be suspected when an intractable diarrhœa exists in a sufferer from pulmonary tuberculosis. Pain is a symptom that is frequently referred to some other part of the body than to the chest. Neuralgic pains may be complained of in almost any part of the body. Headaches and gastralgic pains are not infrequently noted. The shortness of breath from which many of these patients suffer, together with the palpitation of the heart, make many of them think that they are affected with cardiac disease. Involvement of other organs than the lungs in the tubercular process oftentimes gives rise to symptoms that may call attention away from the chest. Thus a chronic or tubercular laryngitis is found. Tubercular or amyloid disease of the kidney may give rise to urinary symptoms, and may cause the appearance of pus, albumin, and casts in the urine.

3. Fibroid Phthisis.

Fibroid phthisis has already been referred to in the section on pneumonia. It is well to remember that it is usually a unilateral disease, often following the inhalation of dust, as in miners and stonecutters. It is essentially a chronic disease, often lasting for years. The physical signs are those of consolidation, with retraction of the chest wall from a contraction of the fibrous tissue which is so abundant in the diseased lung. There is often a distinct depression of the affected side, loss of expansion, dulness upon percussion, though a resonant element may enter into the percussion note, because of the existence of cavities, either pulmonary or bronchiectatic; upon ausculation broncho-vesicular respiration, with moist and dry râles of every variety. The sputa, oftentimes fetid, are usually abundant, especially in the morning, and generally show, upon examination, the tubercle bacillus.

DISEASES OF THE PLEURA.

PLEURISY.

Primary pleurisy is rare. Often, however, we are unable to make out the primary cause during life. It is frequently found in a tubercular nodule of the lung, a tubercular gland, or other tubercular focus. Traumatism, exposure to cold, favor the development, or may be the exciting cause, of pleurisy. In connection with the infectious diseases, particularly rheumatism, pleurisy is not uncommon. Pulmonary diseases, as pneumonia, gangrene, infarction, are commonly accompanied by pleurisy. Disease of the ribs or vertebræ, perforation of the œsophagus, perforation of a gastric ulcer, may induce suppurative pleurisy. Bright's disease is a predisposing cause. Where the exudate is fibrinous and not fluid, the pleurisy is spoken of as fibrinous or dry pleurisy. According to the character of the exudate, we speak of a serous and sero-fibrinous pleurisy, of a suppurative pleurisy or empyema, and of a hemorrhagic pleurisy.

In the majority of cases inflammation of the pleura is a subacute, slow process. Frequently the patient only consults a physician when the effusion has become so great as to embarrass respiration. At times, however, the onset of the disease is marked by quite severe pain in the side, chilly sensations, moderate fever, and dry, hacking cough. Pain, loss of sleep, impaired digestion, cause the patient to look somewhat haggard and pale. When an effusion takes place, the pain is often lessened. The cough of pleurisy is commonly a dry one, unless the disease is complicated by bronchial or pulmonary inflammation. With dry pleurisy the breathing is shallow and rapid. According to the amount of the exudation dyspnœa is slight or well-marked. In many cases orthopnœa is present. The temperature in pleurisy may reach 103°. Its course is atypical. Where there is effusion, even though it is not purulent in character, there may be a slight evening temperature for many weeks. With pus in the pleural cavity, the temperature is usually higher and more irregular than where the effusion is serous, there is much more emaciation, and frequently there are chills and sweats. With pleurisy there may be great muscular weakness, loss of appetite, malaise, emaciation, and constipation. In other cases it is remarkable how the patient will for days or weeks, keep about his work, until finally the difficulty in breathing becomes so great as to force him to seek relief at the hands of a physician. The diminution in the amount of urine, where there is pleural effusion, is sometimes very great. When the effusion is being absorbed, the urine is frequently increased in amount.

Physical Signs. Dry pleurisy has for its characteristic sign the detection of a friction rub upon auscultation. This is most commonly heard just below the nipple on the affected side, and at the point at which the patient locates the pain. It may be very faint, or so loud as to be heard by the patient himself. As to character, it may be squeaking, rubbing, grating, or a fine crackling that resembles closely the alveolar râles. In some cases inspection shows a loss of motion on the affected side.

The physical signs of pleural effusion, where the effusion is large, are as follows:

Inspection. Impairment of motion on the affected side; intercostal spaces bulging, perhaps bulging of the entire side. The hypochondrium may be more prominent because of the downward pressure of the diaphragm. Measurement of the side may show that its circumference exceeds that of the unaffected side. The patient usually lies upon the diseased side, as in this way the weight of the fluid is taken off the sound lung and the heart. Displacement of neighboring organs, as the heart, the liver, can be made out by inspection and by palpation.

Palpation shows a distinct lessening or complete loss of vocal fremitus.

Percussion shows flatness below, gradually merging into dulness and resonance above, as the layer of fluid over the lung becomes thinner and thinner. There is a distinct sense of resistance on percussion over a large amount of fluid. Posteriorly with an effusion of moderate size, the upper boundary of the dulness is highest at the vertebral column, and runs from this point obliquely downward to the side of the chest. Above a pleural effusion there is usually a tympanitic note.

Auscultation reveals a diminution or total loss of the respiratory murmur over the pleuritic effusion. It must not be forgotten, however, that even with a considerable amount of fluid in the pleural cavity, and particularly in children, bronchial breathing can oftentimes be made out. This is a distant bronchial breathing, but is sometimes very deceptive, resembling that of a consolidated lung, or of a cavity. At the upper limit of the effusion friction sounds can occasionally be heard, and the alveolar respiratory sounds often with bronchial râles. A bleating nasal sound, known as ægophony, is sometimes made out as we listen to the voice.

In all cases of doubt, **exploratory puncture** should be made to establish the diagnosis of pleural effusion and the character of the fluid.

Pus in the pleural cavity, constituting the condition known as **empyema**, is usually associated with marked fever of an irregular type, with chills, sweating, emaciation, and oftentimes a typhoid state. The purulent character of the pleurisy is determined by the use of the exploring needle, which must be sufficiently large to allow thick pus to run freely.

A large number of cases of pleurisy with effusion are of tubercular origin. We can suspect the tubercular character when there is a history of hereditary tuberculosis, when we find evidences of tuberculosis in other organs of the body, and when we find the fluid reddened from the presence of blood—hemorrhagic pleurisy. In some cases, though rarely, bacilli have been detected in the effusion.

Differential Diagnosis. The recognition of a large amount of fluid in the pleural cavity is extremely easy, particularly if we remember the use of the exploring needle. At times we are in doubt whether we have to deal with fluid in the chest or with consolidation of the lung, particularly pneumonia. The clinical history is of great value here; in pneumonia the onset is with severe chill, high rise of temperature, cough, rusty sputum. With pleurisy rarely a severe chill, oftener a chilly sensation, fever not so high, cough dry and hacking. Inspection shows more distention of the affected side in pleurisy. Palpation, diminution of vocal fremitus in pleurisy; increase in pneumonia. Percussion often the same in both diseases, but with pleurisy an absolute flatness and a more marked sense of resistance. Auscultation, loss of respiratory murmur or distant bronchial breathing in pleurisy; marked bronchial breathing and bronchophony in pneumonia unless the bronchus be plugged. With pleurisy, too, we have a displacement of the heart, the liver, the diaphragm, which does not occur with pneumonia.

In some cases of intra-thoracic neoplasm or of thickened pleura, the result of preceding pleurisies, the exploring needle is the only means of determining positively whether or not a fluid or a solid gives rise to the dulness and loss of respiratory sounds.

Hydrothorax. The physical signs of hydrothorax are identical with those of pleurisy with effusion. The cause is found in some altered condition of the blood, or some local obstruction to circulation. Most commonly it is met with in connection with Bright's disease, cardiac disease, or in the later stages of the severer anæmias and the cachetic diseases. Frequently, where the cause is general, as in Bright's disease, leukæmia, severe anæmia, the hydrothorax is double. The fluid obtained on aspiration is clear, of low specific gravity, not so rich in albumin and in cellular elements as in pleurisy with effusion. In rare cases blood is present in the pleural cavity, as from traumatism, rupture of an aneurism, in the hemorrhagic diathesis, and in some cases of new growth and of tuberculosis.

PNEUMOTHORAX.

The perforation of the pleura, parietal or visceral, that allows air to enter the pleural cavity from without or through the trachea and bronchi, is usually announced by a sharp pain in the side, soon followed by rapid and difficult breathing, cyanosis, and often pronounced collapse. Death may occur in a few minutes' time, when one lung is already destroyed by tuberculosis, and the comparatively sound lung is suddenly compressed by the air and rendered functionless, or nearly so. Penetrating wounds of the chest wall often cause perforation of the parietal pleura. Tubercular ulceration into the pleura is the commonest intra-thoracic cause. Pleuritic adhesions obliterating the pleural cavity prevent the more frequent occurrence of the accident. Among other causes may be mentioned perforation of the œsophagus, perforation of a gastric ulcer, gangrene of the lung, rupture of a pulmonary abscess. In most of these cases pus escapes into the pleural cavity with the air, or is soon formed as the result of the action of entering pyogenic organisms. Rarely, the accident occurs without sudden symptoms.

The examination of the chest shows in these cases, in addition to the signs of the causal disease, generally tuberculosis, the following: marked bulging and immobility of the affected side, often considerable displacement of neighboring movable organs, as the heart and liver. The patient has an anxious look, is breathing hurriedly, often in the sitting posture. There is cyanosis. Palpation reveals lessened fremitus, and confirms the results of inspection as to loss of motion of the affected side, bulging of intercostal spaces, dislocation of heart and liver. Resonance on percussion is extreme. A deep barrel-note is heard, as over a large air-containing cavity. Auscultation reveals a metallic, amphoric respiration, often almost musical. Light percussion made upon the chest wall, while the auscultator listens with the stethoscope, produces a peculiar metallic reverberating sound, only heard in this condition. Metallic tinkling may be heard if fluid be present as well as air, and if from the wall of the cavity, a drop of fluid falls into the greater body of fluid beneath. So, too, an exaggeration of this sound is obtained by shaking the patient and auscultating. To this metallic splashing sound the term succussion sound has been applied. Where there is an opening into the pleural cavity, allowing both free entrance and exit to air, the bulging of the side will be less marked, the amphoric respiration distinct. Where the opening is partially closed and air is pent up, or where a valve-like flap permits free entrance but not exit of air, bulging and displacement of organs may be extreme, while the amphoric respiratory sounds may be feeble, only heard on inspiration, or lacking. It is, perhaps, needless to note that fluid in the pleural sac would alter the signs. Dulness, the line changing with a change of position of the patient, loss of fremitus, loss of respiratory sounds, would be perceived as in cases of ordinary pleurisy with effusion.

There is often some difficulty in determining whether one has to deal with a pneumothorax or with a large pulmonary cavity. In the latter case, however, there can usually be heard, over or above the amphoric area, vesicular or bronchial sounds; there is not the same displacement of organs, the side is oftener retracted, and the history rarely shows the sudden onset with pain, dyspnœa, cyanosis.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART AND PERICARDIUM.

PHYSICAL EXAMINATION OF THE HEART.

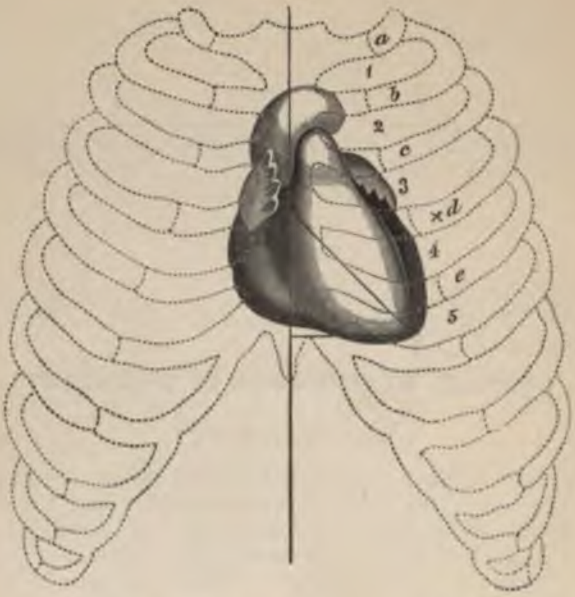
Anatomical Points.

The boundaries of the heart as outlined upon the chest wall, *i. e.*, the boundaries of the præcordia, are: **upper,** the third rib; **lower,** a horizontal line passing through the fifth intercostal space just within the mammillary line, *i. e.*, passing through the site of the apex-beat; **right,** a line parallel to the right sternal margin and a finger's breadth from the same; **left,** a vertical line passing just inside of the left nipple. The apex-beat is normally in the fifth left interspace, a finger's breadth within the **linea mammillaris.**

The portion of the heart not covered by lung is known as the superficial cardiac space, and the portion covered by lung as the deep cardiac space. Absolute dulness on percussion and absence of respiratory sounds mark the limits of the former; relative dulness with superficial respiratory sounds, the latter. The superficial space can, for practical purposes, be represented by a triangle whose base is a horizontal line intersecting the apex-beat and passing through the fifth interspace, whose side is the median line, which, bisecting the sternum, meets the base at right angles, and whose hypothenuse is a line drawn from the apex-beat to a point in the median line, opposite the costal cartilage of the fourth rib.

The normal heart sounds, for whose etiology and characteristics works on physiology may be consulted, are best heard at the following points: Aortic, in the second interspace close to the right margin of the sternum; pulmonary, in the second

FIG. 30.



Superficial cardiac dulness (approximate). (FLINT.)

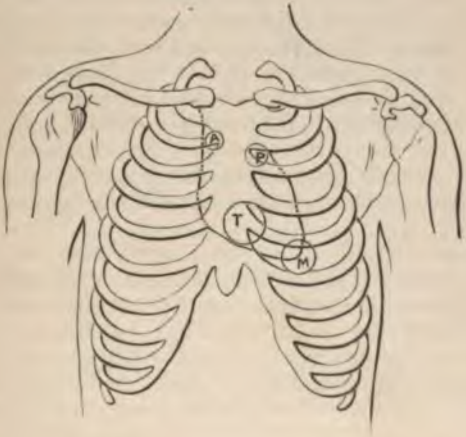
interspace, near the left sternal border; mitral, a trifle to the left of the site of the apex-beat; tricuspid, a little to the right of the inferior border of the heart, usually just above the xiphoid appendix.

While these are the places on the chest wall where the various valvular sounds may best be heard, they do not correspond accurately to the location of the valves. These are located beneath the left edge of the sternum, the pulmonary opposite the second interspace, the aortic opposite the second rib and slightly overlapped by the pulmonary, the mitral at the fourth cartilage, and the tricuspid a little lower down.

Physical Examination of the Heart in Disease.

Inspection of the præcordia is of value in determining the size of the heart. In children particularly, there is often seen a marked bulging of this region where there is pronounced hypertrophy. The apex-beat of an hypertrophic heart is seen further to the left and lower down than normal, often in the sixth or seventh interspace. Even when the hypertrophy is chiefly of the right ventricle, the apex-beat is seen far to the left. With right heart dilatation pulsation of the third, fourth, and fifth interspaces to the left of the sternum, and often epigastric pulsation are common, and where the tricuspid valve is insufficient there is venous pulsation in the neck. A plainly marked, diffuse, heaving, systolic impulse, with its point of greatest intensity to the left of the nipple and in the sixth, seventh or eighth interspace, is sufficient to warrant a presumptive diagnosis of cardiac hypertrophy, from inspection alone. With dilatation the impulse is less clearly seen. Where the apex-beat cannot be seen, hypertrophy is not to be, on this fact alone, excluded. The apex-beat may be concealed beneath a rib. The interposition between the heart and chest wall of lung tissue, as in emphysema, or of fluid, as in pericardial effusion, may also conceal the apex-beat. By inspection, too, the dislocation of the heart by pressure or by traction can be inferred from the location of the apex-beat. When the heart is displaced to the left by pressure, the apex-beat may be

FIG. 31.



Showing præcordial areas. A, aortic area; P, pulmonary area; T, tricuspid area; M, mitral area. (GIBSON and RUSSELL.)

to the left of the mammillary line, but it is also displaced upward, not downward as in hypertrophy. Fluid in the left chest may cause the apex-beat to be seen to the right of the sternum. A pulsation at the apex and another above, especially to the right of the sternum and in the second or third interspace, giving the impression of two apex-beats, is strongly suspicious of aneurism.

A systolic retraction of the præcordial chest wall, particularly the interspaces, is suggestive of adherent pericarditis.

Palpation confirms what has been found on inspection. The location of the apex-beat, the force and rhythm of the heart's action, the area over which the impulse is felt, the retraction or bulging of the præcordial region, are all to be noted by the palpating hand. Not infrequently where there is a pericardial friction rub a fremitus is felt, and with some valvular lesions a thrill is detected, reminding one somewhat of the feeling communicated to the hand laid over the body of a purring cat. It is always best to palpate the præcordia by placing the hand flat upon the chest. This is much better even for locating the apex-beat, than attempting palpation with the finger-tips.

Percussion enables us to outline the size of the heart and the extent of pericardial effusions or growths. Deep cardiac dulness is brought out by rather strong percussion. Commencing far to the right or left of the heart, where is a pure resonant note from the lung, we make percussion, gradually approaching the heart, and mark the point where the first element of dulness, however slight, is first noted. In the same way percussion is made from above downward. By connecting these marks we have an outline of the deep cardiac dulness. This is of great importance in diagnosing cardiac hypertrophy or dilatation, as well as aneurismal enlargements of the vessels at the base of the heart. The superficial cardiac space shows dulness on light percussion and reveals no respiratory sounds on auscultation. In emphysema this space may be obliterated by interposing lung tissue. In pericardial effusion, in hypertrophy, and dilatation, it is increased in extent.

Auscultation. The sounds of the heart may all be practically regarded, so far as diagnosis by auscultation is concerned, as purely valvular, as caused solely by the closure of the valves at the respective orifices. These sounds may be modified or may have added to them new or adventitious characteristics.

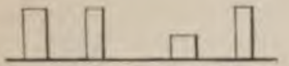
Modifications of the Heart Sounds. These changes are chiefly in intensity and in rhythm.

The **first sound** is of **diminished intensity** from general causes weakening the heart's action, as prolonged fevers, anæmic conditions, cachetic states, or from local conditions. Among the latter may be mentioned the weakening of the cardiac muscle, and consequently the force of the valvular closure, by myocardial degeneration or by dilatation. In fleshy persons, in pericardial effusions, the sound may appear weak because of the layer of fat or fluid interposed between the heart and the examining ear. Here the examination of the pulse may show a stronger heart action than would be recognized from auscultation. Experience alone enables the examiner to say that a heart sound is weak or strong, and it is always to be remembered that there is no absolute standard of intensity of heart sounds or of heart strength, to which all heart sounds can be compared. Each case has to be judged on its own merits.

The **first sound** is of **increased intensity** in states of excitement, at the beginning of acute fevers, in hypertrophy. Particularly is this noted in the hypertrophy consecutive to contracted kidney. Not infrequently the physician's attention is first called to the kidney by his detection of an hypertrophied heart, with heaving impulse and loud, booming first and second sounds, and no valvular lesion to account for these changes. The progress of myocardial degeneration in such cases is usually shown by a gradual lessening of the intensity of the cardiac sounds, by change in rhythm, and by evidences of dilatation.

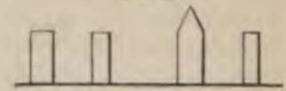
Change of *rhythm* may be due to the loss of a heart-beat here and there, but with some degree of regularity, *e. g.*, the skipping of one beat in five or one in twelve, when it is known as *intermittence*. Or there may be the irregular skipping of beats, often the rapid succession of a few weak beats, then a strong beat or two, or *vice versa*, there being no regularity as to intensity or time. This alteration is spoken of as *irregularity*. And, again, a heart beating regularly may have the first sound made up of two sounds heard at different times, as though the tricuspid and mitral valves did not close synchronously, or as though some valve cusp was late in closing. This change is known as *reduplication*. Clinically we cannot predicate too much upon the detection of reduplication, though it is often found in cases of mitral lesions. An intermittent heart may be normal in its function. Many people always have an intermittent pulse. During fevers, with gastric disturbances, in the neurotic, intermittence is not infrequent. Irregularity is usually of more serious import, denoting marked changes in the nervous or muscular mechanism of the heart. In the profound weakness of fevers, in myocarditis, in fatty heart, and in many cases of valvular lesion where dilatation has occurred, irregularity is pronounced.

FIG. 32.



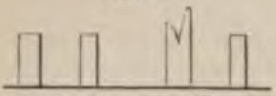
Normal first and second sounds. Diminished first sound.

FIG. 33.



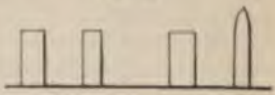
Normal first and second sounds. Accentuated first sound.

FIG. 34.



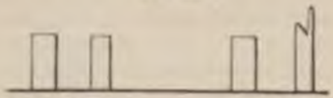
a. Normal first and second sounds. b. Reduplicated first sound.

FIG. 35.



Normal first and second sounds. Accentuated second sound.

FIG. 36.



a. Normal first and second sounds. b. Reduplicated and accentuated second sound.

Illustrating diagramatically modifications of the heart sounds (GIBSON and RUSSELL.)

The **second sound is less intense** in cases where the ventricular contraction is weak, as in fatty heart, myocarditis, dilatation, or where a diminished volume of blood is thrown into the aorta, as in acute anæmia.

When obstruction to the general circulation exists, as in arterio-sclerosis, contracted kidney, there is an **accentuation** of the aortic **second sound.** The pulmonic second sound, which is normally fainter than the aortic, may, where there is obstruction to the pulmonic circulation, as in emphysema, some cases of phthisis, and mitral disease, be distinctly louder and more accentuated than the aortic. The comparison is very easily made, as both sounds are best heard in the second interspace, the one just to the right, the other to the left of the sternum.

Alterations in **rhythm**, as reduplication, intermittence, irregularity, may occur with the second sound as with the first.

The **adventitious sounds** of the heart are abnormal and are usually spoken of as murmurs. These may be exocardial or endocardial.

The **exocardial sounds** are pericardial. At the beginning of pericarditis, and again when an exudate has been absorbed, the roughened visceral and parietal layers of the pericardium, that normally move noiselessly over each other, give forth a friction rub, that is, creaking or rubbing in character, and resembles the sound of the pleural friction rub. This is best heard near the base, is synchronous with the heart beat and not with the respiratory movements, and is often intensified by pressure with the stethoscope or by having the patient lean forward.

In pleuritis with pericarditis a friction rub is heard, caused by the rubbing together of roughened pleura and pericardium. This is usually heard with the systole or during inspiration, by which fact it can generally be distinguished from a pure pericardial friction sound, though at times the differential diagnosis is impossible.

A **succussion sound** is heard where air and fluid are present in the pericardial sac, a rare occurrence.

In diagnosing a valvular lesion by the presence of a murmur three questions are to be answered: First, at **what point** is the murmur heard with greatest intensity? Second, with **which sound** of the heart is it heard? *i. e.*, is it systolic or diastolic? Third, in **what direction** is it transmitted? Aid is also derived from (1) the character of the pulse, (2) by observing the part of the heart that is hypertrophied in consequence of the valvular lesion, as well as by (3) the history of the case. The student should in each case picture clearly to himself what is the physiological action of the heart at the point and time of observation, should have a clear conception of what is the normal direction of the blood current, and what is the normal function of the valve he is examining. In this way he can reason out what must be the character of the lesion producing the abnormal sound.

When, in consequence of disease, the valve is covered with an excrescence, as in benignant endocarditis, or when there is loss of tissue, inflammatory infiltration and swelling, perhaps perforation of a valve, as in malignant endocarditis, or when, through chronic inflammatory or sclerotic changes, a valve becomes thickened, deformed, shrunken, adherent to its neighbor valve, perhaps calcareous, it is easily understood how such a valve may close imperfectly, may allow blood to escape when closure should be perfect, how it may leak or be incompetent; or how, on the contrary, the valve, stiff and adherent, may fail to open as widely as it should, thus presenting an obstacle to the free passage of the blood current through it. The former condition, producing a leak in the valve, is known as a regurgitant lesion; the murmur it causes as a regurgitant murmur or as the murmur of valvular insufficiency or incompetency. The latter condition, where there is an obstacle to the flow of blood, is known as an obstructive lesion, the murmur as an obstructive murmur.

Let us take, as an example, a narrowing and partial closure of the aortic orifice. As the blood is forced through this narrow orifice a murmur is produced. But blood passes normally from the left ventricle through this opening with the systole, *i. e.*, at the time of the first sound; therefore, we shall hear an aortic obstructive murmur synchronously with the apex impulse. It will be best heard in the aortic area, at the base of the heart. Listening, therefore, in the second interspace near the right sternal margin, we heard the murmur loudest. So, too, the sound transmitted in the direction of the current is heard in the large vessels of the neck, perhaps even in the brachial and femoral arteries. We may say, then, that when a murmur is systolic, best heard in the right second interspace close to the sternum, and transmitted along the course of the large vessels, it is due to aortic obstruction. It is well to refer here to the fact that this murmur—and what is said of this murmur applies in a measure to all—when loud, may be heard over a wider area than the "aortic area." Thus it may be heard at the apex and simulate mitral regurgitation; and particularly, the sound being well conducted by the sternum, it is often clearly heard over this entire bone. The diagnosis of the valve affected is to be made not on the point where the murmur is heard, but where it is **best** heard, *i. e.*, the point of **maximum intensity**, in this particular instance the base of the heart, over the right sternal border opposite the second interspace or the second costal cartilage.

Where the aortic valve is incompetent and leaks, the murmur is best heard a little lower down, near the left edge of

Often best heard in Mitral area

the sternum, opposite the third interspace. It is frequently clearly transmitted throughout the entire length of the sternum even to the xiphoid appendix. In time it is diastolic, often completely replacing the aortic second sound.

Mitral murmurs are best heard near the apex. A **mitral regurgitant** murmur, the commonest of all, is best heard over the apex or a little to the left of the apex, is systolic and transmitted to the left, sometimes being plainly heard even in the axillary space or near the inferior angle of the scapula. The first sound of the heart is at times almost completely lost in this murmur.

The murmur of **mitral obstruction** is best heard just above the apex-beat. This murmur is not clearly transmitted in any direction, being more localized than any other murmur. As to time, it is diastolic, or often, more strictly speaking, presystolic, heard just before the first sound, into which it seems to merge, or by which it is suddenly cut short.

As the left heart is much more frequently the seat of valvular lesions than the right heart, save in intra-uterine life, these murmurs already considered, of the aortic and mitral valves, are the ones commonly met with.

A **pulmonary obstructive murmur** is systolic, best heard in the second left interspace, close to the sternum. Unless very loud it is not widely transmitted. Congenital narrowing of the orifice is the commonest cause of this murmur. Systolic pulmonary murmurs are often anæmic.

Pulmonary regurgitation is rarely met with, and presents nearly the same auscultatory phenomena as aortic regurgitation. The murmur is diastolic, transmitted downward, along the sternum, the point of maximum intensity being in the sternum opposite the third cartilage. Hypertrophy of the right rather than the left ventricle, and the hearing of the murmur more clearly over the right than over the left ventricle, would favor the diagnosis of pulmonary rather than aortic regurgitation.

Tricupid obstruction is rare. A diastolic or presystolic murmur heard best over the sternum just above the xiphoid cartilage, and transmitted downward and to the left, would lead to the suspicion of an obstruction at this orifice.

Tricuspid regurgitation is much commoner, being often heard when the right chamber dilates and the valve becomes "relatively insufficient." The backward current of blood from the right ventricle into the right auricle causes a systolic murmur, best heard over the lower half of the sternum. At times it can be heard transmitted to the right. As a confirmatory evidence of a tricuspid lesion allowing of regurgitation, is to be mentioned a systolic pulsation of the veins of the neck.

Simple murmurs are usually recognized without much difficulty. Combinations of murmurs, however, are common, and may offer great obstacles to their recognition, or, as sometimes happens, it may be impossible accurately to define the nature of the murmur, and therefore of the lesion. This is particularly true where two murmurs coexist, both systolic, as, for instance, an aortic stenotic, and mitral regurgitant, and where each murmur is loud and heard over the entire præcordial region. If cardiac irregularity exists at the same time it adds to the difficulty of diagnosis.

Yet, by carefully studying the sounds heard, one can usually differentiate between the murmurs. Thus, in the case in question, the aortic stenotic murmur is heard loudest over the manubrium and is transmitted into the carotids. Now, as we auscultate downward toward the apex, we notice, first, that the systolic murmur grows fainter until we reach a point near the apex, where it again increases in loudness, *i. e.*, where the sound of the mitral systolic murmur is heard. We also note that the systolic murmur is transmitted to the left, another evidence of its being mitral. In many cases, too, there is a difference in quality or in pitch that serves to indicate that there is more than one lesion. The one sound may be loud and rough, as is often the case in aortic stenosis: it may even be musical in character, while the other, as frequently in regurgitation, may be soft and blowing.

Points of greatest intensity of murmurs, variations in quality and pitch, are therefore to be noted when one is attempting to analyze a compound murmur. Other facts in the clinical history and in the physical examination may help, which will be referred to later. Thus, in mitral stenosis the irregularity of the pulse, the dilatation of the right heart, the accentuated pulmonic second sound may aid in establishing the diagnosis. In tricuspid regurgitation, pulsation of the cervical veins may give a clew to the lesions, as a "water-hammer" pulse may to aortic regurgitation.

Corrigan's pulse - jerky

There are a few general facts that, if kept in mind, will be of great service. (*a*) Far oftener a primary extra-uterine lesion is in the left than in the right chamber of the heart; a congenital lesion in the right heart. (*b*) Murmurs may change their character from day to day, as in acute endocarditis and in chronic disease where the cardiac force varies. Thus, toward the close of life, or with great weakness of the cardiac contraction, a murmur, as of mitral stenosis, may grow very faint, or may totally disappear. (*c*) Valvular disease may exist without a murmur, as in the case of a very feeble heart, and *per contra* a murmur may exist and no valvular disease be present, as in the anæmias. Thus too much stress is not to be laid upon the presence or absence of cardiac murmur. Other evidences of disease, *e. g.*, hypertrophy, dilatation, arrhythmia, stasis, etc., must be sought for before a diagnosis of valvular disease is positively made.

MITRAL REGURGITATION OR INSUFFICIENCY.

Thickening of the mitral cusps, contraction and deformity of their free edges, shortening of the cordæ tendineæ, may allow blood at each systole to escape back into the left auricle, which soon, from overdistention and increased work, becomes dilated and hypertrophied; the left ventricle also, receiving an increased amount of blood, dilates, and from the increase in work necessary to expel the extra amount of blood, hypertrophies. The backing up of the blood causes stasis in the pulmonic circulation, increased work of the right heart, and consequent hypertrophy. Later, as in all forms of valvular disease, general venous stasis may occur.

The physical signs of this lesion are as follows:

Inspection. In children, prominence of præcordial region from the yielding of the thoracic wall to the hypertrophied heart. Diffuse pulsation, apex-beat to the left and downward. With failure of compensation, epigastric pulsation and general venous stasis.

Palpation. Apex-beat strong, displaced to the left and downward, systolic thrill at times. Pulse usually regular, fairly strong.

Percussion. Area of cardiac dulness increased to the left; later to the right as well.

Auscultation. Apical, systolic, loud, long murmur transmitted to the left; may entirely replace the first sound. Pulmonic second sound accentuated.

MITRAL OBSTRUCTION OR STENOSIS.

This lesion is often found in combination with, or as a sequel to, mitral insufficiency. The narrowing of the mitral orifice necessitates increased work on the part of the left auricle, which hypertrophies, dams up the blood in the auricle and pulmonary circulation so that very early right-heart dilatation and hypertrophy ensue, often with a relative insufficiency of the tricuspid valve and evidences of general stasis. The left ventricle and the arterial vessels receive but a limited supply of blood through the contracted mitral opening. The left ventricle is, therefore, small, the arteries are poorly filled, and the pulse is small. Often it is weak, irregular, and rapid.

Physical examination reveals the following:

Inspection. Pulsation in epigastrium or jugulars from right-heart enlargement. Apex-beat feeble, though perhaps pushed to the left by the enlarged right heart.

Palpation. Epigastric pulsation at times, diastolic thrill. Pulse small, rapid, irregular.

Percussion. Heart's dulness is increased so that it may pass right sternal border; increased to the left because the left ventricle is pushed to the left by the enlarged right ventricle. The left ventricle is often hypertrophied because of a coexisting mitral insufficiency.

Auscultation. A bubbling or rippling murmur is heard just before the first sound, which is often unnaturally loud and accentuated, and which sharply cuts short the murmur, if no insufficiency coexists, *i. e.*, a presystolic murmur. This murmur may fill the time of the entire period of rest of the heart, *i. e.*, may be diastolic. It is distinctly localized at a point just above the apex-beat, very rarely transmitted.

The second sound at the base is often reduplicated and the pulmonic sound accentuated. Subjective symptoms, dropsical manifestations, are commonly well marked in this lesion.

AORTIC INSUFFICIENCY OR REGURGITATION.

When the aortic valve closes imperfectly the left ventricle receives, during diastole, blood from the left auricle as well as the blood returned through the leaking valve from the aorta. Overdistended and overworked, the ventricle dilates and hypertrophies and often reaches an enormous size, throwing with great force a large quantity of blood into the aorta and systemic circulation, suddenly distending the bloodvessels, which as suddenly collapse as some of the blood hurries on into the capillaries, and another large part falls back into the ventricle through the faulty valve.

Inspection. Often pallor of the face. Prominent præcordial region. Apex-beat strong, diffuse, heaving, situated to the left and downward. Peripheral arteries, as the temporal, brachial, femoral, pulsate strongly and are often tortuous. Capillary pulse often seen. This pulse, first described by Quincke, is seen best under the finger-nails, where with the systole there is marked redness, with the diastole pallor, due to the sudden filling and emptying of the capillaries. In an hyperæmic line, drawn on the forehead by a stroke with the finger-nail, the same alternate blushing and pallor can be seen.

Palpation. Strong, heaving, displaced apex-beat. At times the heaving pulsation of the entire left heart is seen communicated to the chest wall. Radial pulse, strong, with a quick, jumping, springing, water-hammer stroke, and a very rapid subsidence (*pulsus celer*, Corrigan's pulse, water-hammer pulse). Even in the smaller arteries the pulse has this peculiar quality.

Percussion. Increased area of cardiac dulness to the left, downward, and upward. At times dulness over the dilated aorta can be made out. This may simulate aneurism.

Auscultation. Loud, blowing, diastolic murmur, best heard at the left sternal border, about opposite the third or fourth interspace. Often heard over entire cardiac region. Over the femoral and brachial arteries and at times over the more peripherally located, a sharp, tense, impulsive stroke is heard by the stethoscope, resembling an accentuated pulmonic second sound. Pressure with the instrument changes this to a loud systolic murmur. A double sound is sometimes heard in the femoral artery. The exact explanation of these sounds in the peripheral arteries has not yet been very satisfactorily given.

While aortic insufficiency is one of the severest forms of the disease to treat when once compensation begins to fail, it is remarkable in how many cases subjective symptoms are absent until very late, as Nature's compensation is for a long time so nearly perfect.

AORTIC STENOSIS. AORTIC OBSTRUCTION.

This lesion is not infrequently found in combination with aortic insufficiency. As a solitary valvular lesion it is rare. Dilatation and hypertrophy of the left ventricle with subsequent similar changes in the right heart are the necessary consequences of the lesion.

On examination there is found the following:

Inspection. Apex-beat diffuse, sometimes slow, to the left.

Palpation. Confirmatory of results of inspection. Apex-beat often rather weak. Pulse small and often slow.

Percussion. Increased area of left-heart dulness.

Auscultation. Long, loud, sawing, or musical, systolic murmur. This is best heard over the right edge of the manubrium and in the second right interspace close to the sternum. The murmur is propagated into the large vessels of the neck.

With severe grades of aortic stenosis the prognosis is grave. Not only does right heart dilatation occur early, with failure of compensation, but there is often pain, tinnitus, vertigo, attacks of syncope, and even convulsions of an epileptiform character.

TRICUSPID INSUFFICIENCY.

In extra-uterine life insufficiency of the tricuspid valve is rare, except as an affection secondary to valvular disease of the left heart, a secondary endocarditis, or the so-called "relative insufficiency," where the valves fail to meet because of excessive dilatation of the cavity. Malignant endocarditis does, however, at times primarily affect the valves of the right heart, and under such circumstances regurgitation might occur at the tricuspid orifice.

Enlargement and hypertrophy of the right ventricle ensue as a consequence of the lesion under consideration. Also when the systolic blood is forced back into the right auricle and into the large veins. A systolic pulsation is thus seen in the jugulars, extending to the jugular valve, or if this gives way, as it often does, throughout the entire length of the jugulars, particularly the right one. In advanced cases, where there is a strong systolic beat, the regurgitation is sufficient to cause systolic distention of the hepatic veins and their rootlets, and we have the phenomena of the "hepatic venous pulse."

Auscultation reveals a murmur over the sternum, at its lower end, closely simulating a mitral systolic murmur, and, when the latter murmur coexists with the tricuspid murmur, very difficult to distinguish from it. With evidences by inspection, palpation, and percussion, of right-heart dilatation, of a venous pulse, and also the proof of a primary lesion in the left heart, to account for a relative insufficiency, we can, when this murmur is detected, usually safely diagnose regurgitation at the tricuspid orifice.

TRICUSPID OBSTRUCTION OR STENOSIS.

Tricuspid stenosis is an exceedingly rare form of valvular disease, seldom met with except as a congenital malformation, or as a lesion secondary to lesions of the left heart, usually mitral stenosis. The physical signs will be those of dilatation of the right auricle with a diastolic murmur best heard over the tricuspid area. Evidences of venous stasis would be early and marked.

PULMONARY INSUFFICIENCY OR REGURGITATION.

Pulmonary insufficiency is met with as a congenital malformation, usually in connection with other cardiac defects. The physical signs would be those of dilatation and hypertrophy of the right heart, and a loud murmur best heard to the left of the sternum in the second intercostal space, diastolic as to time, and transmitted downward over the sternum. In this form of valvular disease, when there is marked hypertrophy of the right ventricle, compensation may be quite perfect for a long time.

PULMONARY STENOSIS.

Stenosis of the pulmonary orifice is the commonest of the congenital forms of heart disease, whether its cause be looked upon as an intra-uterine endocarditis, or as a failure in development. It is usually associated with other developmental defects in the heart, such as defects in the ventricular septum, patency of the foramen ovale, etc. Symptomatically pulmonary stenosis is noted soon after the birth of the child. Children thus affected are popularly known as "blue babies" because of their marked cyanotic appearance. The cyanosis is most plainly seen when the child cries or becomes nervously excited, or after violent exertion. Many children with pulmonary stenosis and the other defects of development already mentioned, live for but a few days. Others may live five, ten, or even twenty years.

The physical examination of such a child on **inspection** reveals the following: The face, lips, hands, and nail-beds are cyanotic. The nails are often curved in a claw-like manner, and the ends of the fingers club-shaped. The child may show failure of development in its bony and muscular structure. The cardiac region is usually prominent. Epigastric pulsation may be noted. **Percussion.** Increased area of cardiac dulness, especially to the right. **Auscultation.** A systolic murmur is heard, best at the second left intercostal space. This murmur is often very loud, and clearly heard over the entire cardiac region. It is very difficult in these cases to say positively, as the result of physical examination, whether a stenosis of the pulmonary valves alone is present, or whether there is also a patent foramen ovale, a persistent ductus Botalli, or a defect in the ventricular septum. It can usually be assumed that where there is an obstruction at the pulmonary valve, or in the pulmonary artery, or in the conus arteriosus, that the foramen ovale and the ductus Botalli are patent. Symptoms and signs of general stasis sooner or later become very marked in cases of congenital lesions of the heart, and the outlook is, almost without exception, grave.

GENERAL SYMPTOMATOLOGY AND SEQUELÆ OF VALVULAR DISEASE OF THE HEART.

It is astonishing how many patients suffering from valvular disease of the heart consult a physician for symptoms that, to the patient at least, are indicative of disease in some other organ than the one really affected. A valvular disease of the heart where compensation is perfect may exist for many years without causing any unpleasant symptoms; then, as compensation begins to fail, symptoms are first manifested, perhaps in organs somewhat distant from the heart. It may be that a hyperæmic liver causes, by stretching of the capsule, pain in the hypochondrium; or from passive congestion of the blood-vessels of the stomach, symptoms of indigestion, as nausea, vomiting, or of intestinal disturbance are manifested. The shortness of breath and the hacking cough may lead the patient to think that the lungs are affected. This should put the physician upon his guard, and he should always examine the heart, even though the symptoms of the patient do not point directly to that organ.

Perhaps the commonest complaint of patients with valvular disease, where compensation fails, is of **shortness of breath.** Dyspnœa comes on after severe exertion at first, but soon the patient notices that even climbing of a few steps or running a short distance, causes great difficulty in breathing.

Associated with this is often a sense of fluttering of the heart —**palpitation** of the heart. When the heart is acting naturally the patient is unconscious of its beat. Why a patient with a diseased heart should be conscious of its beating we do not know, but it is a fact that with failure of compensation palpitation is frequently complained of, and where the heart is hypertrophied and beats violently and rapidly, the patient often feels the impulse against the chest wall, and at times can hear the sounds of the heart. In some cases of valvular disease the palpitation of the heart is paroxysmal, the heart, during a paroxysm, going with great rapidity, the patient being conscious of the heart's action, and suffering intense anxiety and a sense of oppression. To this exceedingly rapid palpitation of the heart the name **tachycardia** is given.

Cough is present where the pulmonary vessels are congested and where there is a slight transudation from them into the alveoli or bronchi. The cough is at first merely a dry, hacking, irritating cough ; later it may be accompanied by a slight, frothy expectoration, and, as compensation fails more and more, the expectorate may become more profuse and tinged with blood.

Pain is frequently absent throughout the entire course of valvular disease of the heart. It is commoner in stenosis of valves than in insufficiency of valves. Particularly is this true of aortic disease. In some cases, however, pain in the region of the heart is severe. At times there is a sense of oppression, and, particularly when the valvular disease is associated with sclerosis of the coronary arteries, pain may be of the most excruciating character, coming on in severe paroxysms, as an attack of **angina pectoris.** During the course of heart disease pain in other organs may be produced as stasis becomes marked. Thus a distended liver, spleen, or kidney may be painful and tender. In aortic disease headache is not uncommon.

Pulse. The pulse of valvular disease is usually an irritable one, easily excited to rapid action. The average rate with valvular disease is high, though in aortic stenosis the pulse is frequently slower than normal. In many cases the pulse is irregular. This is particularly true in cases of mitral stenosis, and intermittence of the pulse is also noted at times. The peculiar pulse of aortic insufficiency has been described.

Nervous Symptoms. In valvular disease nervous symptoms are often very slightly marked, if at all. Many patients, however, become somewhat irritable, fretful, even melancholic. Disturbed circulation in the brain may give symptoms of dizziness, of tinnitus aurium, of headache. Severe lesions of the brain may be produced by cerebral embolism, or by the rupture of a diseased vessel under high arterial pressure. Pains in the arms and in the joints are at times met with. The association of aortic disease with other sclerotic changes should be remembered. Numerous cases of the combination with locomotor ataxia are on record.

Embolic Symptoms. From the diseased valves, or from thrombotic masses in a distended heart, particularly in the auricles, fragments may be detached, and, carried into the circulation as emboli, lodge in various organs of the body, and produce a variety of symptoms according to the organ affected and the importance and size of the bloodvessel. From the right heart, emboli may lodge in the lung, producing the symptoms of pulmonary infarction. The obstruction of the cerebral arteries with the phenomena of an apopletic seizure, have already been referred to. An embolism of the kidney may manifest itself by pain in the region of the kidney and by hæmaturia. Post-mortem infarcts in the spleen are commonly found. At times during life they may be suspected by the occurrence of sudden pain in the region of the spleen. In the mesenteric vessels emboli are rare, and yet they are occasionally met with, giving the symptoms of pain and collapse as from severe hemorrhage. In the peripheral arteries as, for instance, the femoral, the embolus produces pain, swelling of the affected limb, and, unless collateral circulation be sufficient, gangrene of the part.

Stasis. When compensation fails in the case of lesions of the left heart, the lungs soon become congested, and this is manifested by the dyspnœa, the cough, the watery, frothy, blood-tinged sputa. The patients also become cyanotic, the bluish tint being especially noticeable on the lips and under the nails. When there is failure of the right heart the general venous system is overdistended with blood, transudations occur in various organs of the body, and we have the appearance of general œdema, or anasarca. The liver is enlarged and painful and there may be slight jaundice; the kidneys become congested, the urine is scanty, of high specific gravity, and often albuminous. A large painful spleen can be made out upon percussion or even upon palpation. Congestion of the blood-vessels of the stomach and of the intestine causes the symptoms of a gastro-enteric catarrh, derangement of the appetite, vomiting, diarrhœa. The œdema of the subcutaneous tissues is usually first noted where the tissues are loose and in the dependent portions of the body. Thus, at night a swelling may be noted about the ankles; in the morning beneath the eyelids there is a puffiness. As the venous congestion becomes more marked, the swelling increases in extent, does not disappear when the part is elevated, and we may finally have the patient with the limbs immensely swollen, with transudates in all the serous cavities—hydroperitoneum, hydrothorax, hydropericardium—with the face and even the arms and hands greatly swollen, presenting much the same picture as is presented by a patient with chronic parenchymatous nephritis.

Many patients with valvular disease of the heart are well nourished even to the end. This is particularly true where the patient has a good digestion and where the disease does not disturb his sleep too much. In other cases the digestion is much impaired, sleep is disturbed, the patients become anæmic, the functions of the kidneys are imperfectly performed, and we find the patient pale, weak, emaciated, as in a severe wasting disease.

Recurrences and exacerbations of valvular disease are not uncommon. Fresh inflammatory action is excited on the valves of the heart, and we may have all the evidences of an acute or subacute endocarditis added to those of the chronic valvular lesion. Many patients by care and caution are enabled to do light or even quite heavy work for months; then compensation seems to fail. Under rest and cardiac stimulants the heart is again restored in vigor, and the patient is able to work for months. Many expect once or twice a year to lay up for repairs in the hospital.

The following table, modified after Strümpell, gives in concise form the physical signs of valvular disease of the left heart, *i. e.*, of the commonest valvular lesions:

COMPARATIVE TABLE OF THE MOST IMPORTANT PHYSICAL SIGNS IN VALVULAR DISEASE OF THE HEART.

Form of heart disease.	Inspection.	Palpation.	Percussion.	Auscultation.
1. Mitral insufficiency.	Apex-beat strong, displaced downward and to the left.	Systolic thrill at apex; radial pulse nearly normal.	Hypertrophy of the left, later of the right, ventricle.	Systolic, apical murmur transmitted to the left; pulmonic second sound accentuated.
2. Mitral stenosis.	Cardiac impulse diffuse; epigastric pulsation common; pulsation of cervical veins from tricuspid relative insufficiency.	Diastolic thrill at apex; pulse small, irregular, rapid.	Hypertrophy of right ventricle; usually also of left from accompanying mitral insufficiency.	Diastolic or presystolic apical murmur, not transmitted; accentuated first and pulmonic second sounds; often with very weak heart, *e. g.*, just before death, murmur disappears.
3. Aortic insufficiency.	Apex-beat strong, to the left and downward; peripheral arteries tortuous and pulsating visibly; often capillary pulse.	Strong, heaving apex-beat. *Pulsus celer.*	Hypertrophy of left ventricle.	Diastolic murmur over upper sternum; characteristic sounds in arteries (brachial, femoral, etc.).
4. Aortic stenosis	Apex-beat to the left.	Heart's action not very strong; pulse slow.	Hypertrophy of left ventricle.	Loud systolic murmur in second right interspace transmitted into large vessels.

ACUTE ENDOCARDITIS.

Acute endocarditis may affect the mural endocardium, but much oftener the endocardium of the valves. This disease is excited by micro-organisms or by their products. It is commonly divided clinically into the benignant or verrucous endocarditis, and the malignant or ulcerative endocarditis, as it is found post-mortem, in the so-called benignant cases, that the valves are covered, particularly at their edges, with an elevated warty vegetation, while in the malignant forms there is a destruction of tissue, a true ulceration. It is very difficult or even impossible, clinically, to draw a clearly defined line of distinction between benignant and malignant cases, and it is also difficult or impossible, at times, to say post-mortem, that a case of endocarditis is benignant or malignant, making the distinction solely upon the absence or presence of ulcerations. Not a few cases of malignant endocarditis, fatal through sepsis and not through mechanical disturbances of circulation, are found, post-mortem, to have no anatomical ulcer upon the valves or upon the mural endocardium.

Acute endocarditis as a primary affection is rare. Almost always the valves are affected secondarily. In the benignant form of the disease rheumatism is the primary affection in the majority of cases; other acute infectious diseases, as, for instance, pneumonia, typhoid fever, scarlet fever, may, however, have acute endocarditis as a complication. In the benignant form of the disease micro-organisms are usually found upon the valves, but no specific organism has yet been identified as the sole cause of the acute valvular inflammation. The malignant form of endocarditis is produced usually by the pyogenic organisms. The diplococcus of pneumonia, the gonococcus, the bacillus of diphtheria have been found, as well as the streptococcus and staphylococcus pyogenes. The primary focus of the disease is oftentimes discovered in a wound of the surface of the body, in a gonorrhœa, in a diphtheria, in an infected post-partum uterus, or it may be found in a suppurative process in some internal organ, as the middle ear, the gall-ducts, an appendicular abscess.

ACUTE BENIGNANT ENDOCARDITIS.

Acute benignant endocarditis is not infrequently overlooked, as no symptoms call attention to the heart. During the course of an acute rheumatism, as well as during the course of any other infectious disease, the heart should be carefully examined, particularly to see if murmurs develop. When the valves are affected, murmurs in the left heart are almost sure to be heard. It is not always easy to differentiate between the murmur of an acute endocarditis occurring during an infectious disease, and the hæmic murmurs so frequently found, but the hæmic murmurs are always systolic and heard most commonly over the pulmonary area, and they disappear with the disappearance of the acute disease. The murmurs of acute endocarditis, on the contrary, may be systolic or diastolic, are very rarely in the right heart, and are permanent.

Symptoms sometimes direct attention to the heart. There may be slight pain or discomfort, not infrequently a feeling of oppression. The heart's action is often increased in rapidity; the pulse may be intermittent. A slight rise in temperature may mark the onset of acute endocarditis, though the additional temperature is usually overlooked, as the affection comes on during the course of a disease where fever is already a symptom. Following the acute affection there are usually the changes incident to chronic valvular disease. If the mechanical interference with the circulation is great, dilatation and hypertrophy and failure of compensation may come on rapidly; in other cases it is months or years before any symptoms of compensatory failure are manifested.

Some forms of acute endocarditis, running a benignant course, have symptoms of a much graver character than those already described. These are particularly the recurrent forms of the disease. Often these are exacerbations of an old valvular endocarditis. Here a sense of oppression in the chest, dyspnœa, irritable heart's action, rise in temperature, with evidences of moderate sepsis, often with general malaise, headache, and even slight chills, mark a disease that approaches in its clinical picture the form of endocarditis known as malignant, and yet that must be classed clinically as benignant, because of its favorable outcome. In the benignant forms of the disease, embolic symptoms may be present as in the malignant form, but here the embolus is aseptic or comparatively so, and we have, as a consequence of its lodgment in some artery, merely the mechanical effect of the plugging of the vessel; rarely do we find suppurative processes excited at the point of lodgment of the embolus.

MALIGNANT, SEPTIC, OR ULCERATIVE ENDOCARDITIS.

Malignant, septic, or ulcerative endocarditis occurs oftenest in the form of a severe infectious disease closely resembling pyæmia, but in which the heart seems to play the most important part, and from this fact gives its name to the malady. The patient may be stricken down quite suddenly with a high fever and with chills. The chills are usually severe and irregular, resembling exactly those of pyæmia. The lodgment of septic emboli in various organs of the body may give rise to multiple abscesses, so that we may strictly say that the process is pyæmic. The temperature, as in pyæmia, is very irregular, at the time of the chill often reaching a height of 105°–106°, at other times being, perhaps, normal. In some forms of the disease there is such marked remission and such a periodicity to the chills, fever, and sweatings as to lead one to suspect malaria. In any such case the blood should be examined for the plasmodium, and quinine should be given for diagnostic purposes.

The general appearance of a patient with malignant endocarditis is that of a very sick person. He is soon in the typhoid state, becomes weak, emaciated, has a foul, dry, cracked tongue, a disordered stomach and bowels, perhaps involuntary discharges, as in any severe infectious disease. Delirium may be pronounced.

The resemblance to typhoid fever is in some cases very great, and particularly where the chills are not severe, and where the onset of the disease and its progress are rather slow. In these cases, too, the occurrence of an eruption makes the simulation more perfect. The eruption in malignant endocarditis is due to minute cutaneous emboli; but it is to be noted of this eruption that the color does not disappear so readily on pressure as in the eruption of typhoid; that the spots are more universal; and also that in the centre of each spot there is an anæmic area that is not seen in the spots of typhoid fever. (Fræntzel.) These spots, too, may occur earlier in the course of the disease than in typhoid fever, that is, before the seventh day. The embolic symptoms in malignant endocarditis may be pronounced. Embolism of the kidney, spleen, liver, brain, and of the peripheral arteries will present the same symptoms as in the benignant form of endocarditis, except that the embolus, being septic, may excite suppurative processes.

In malignant endocarditis retinal hemorrhages are of great value in establishing a diagnosis, as they are not found in the benignant form of the disease, nor in malaria or typhoid fever.

The heart, when examined, usually gives us a clew to the diagnosis. Murmurs are distinctly heard, and they are often very changeable in character from day to day. These murmurs are almost always in the left heart, though the malignant form of endocarditis attacks, oftener than is generally supposed, the valves of the right heart.

It is worthy of note, too, that on valves already sclerotic, organisms may become implanted and excite a severe and septic form of the disease, so that we may have a patient, a sufferer from a chronic valvular disease, suddenly exhibit the symptoms of a severe infectious disease that runs a fatal course, and where death is due, not to the mechanical interference with the circulation, but really to septicæmia.

Differential Diagnosis. **Malaria** is recognized by a knowledge of exposure, the presence of the plasmodium in the blood, and the abatement of fever and chills on the administration of quinine.

From **pyæmia** it is not to be distinguished, as it is really in such cases but a part and parcel of this disease.

The differential diagnosis between malignant endocarditis and **typhoid** is oftentimes extremely difficult, particularly in those cases of endocarditis where the disease commences somewhat slowly, with the vague, indefinite symptoms of headache, malaise, loss of appetite, and where the temperature has a distinctly remittent character, where the bowels are loose, where there is tympany, and where the nervous and mental symptoms are marked. The main facts that enable us to make a differential diagnosis are the following:

1. In endocarditis there is usually a history of infection, a suppurating wound, an infected uterus, or an internal abscess.
2. The rash of endocarditis may appear early, is not so apt to be limited to the abdomen and chest, and may be ecchymotic.
3. Symptoms of emboli in the kidney, spleen, brain, and other organs are much commoner in endocarditis than in typhoid.
4. Retinal hemorrhages seldom occur in typhoid, but are common in endocarditis.
5. Hæmatogenous jaundice is not infrequent in endocarditis.
6. Ehrlich's reaction is common in typhoid, rare in endocarditis.
7. Erythematous rashes are common in endocarditis, rare in typhoid.
8. The pulse of endocarditis is frequently rapid, irregular, varies much from hour to hour or from day to day; the same may be said of the respiration. In typhoid the pulse is slow as compared to the temperature.
9. The heart in endocarditis sooner or later reveals a murmur at the mitral or aortic valve, more rarely at the valves of the right heart. The murmur in typhoid is in almost every instance a hæmic murmur.
10. Pus-producing organisms can oftentimes be proven in the blood of endocarditis. The typhoid bacillus is found in the blood and stools of typhoid fever.

MYOCARDITIS.

Myocarditis may be an acute circumscribed or an acute diffuse inflammation of the myocardium; in the former case an abscess of the heart-wall, in the latter multiple abscesses, as in pyæmia. Such cases are very rare and extremely difficult to diagnose with certainty.

Chronic myocarditis may be more or less circumscribed or diffuse. It is usually the indurative or cicatricial process that follows the necrosis of a limited area of the heart muscle from the blocking up of a coronary artery or one of its branches by a thrombus, an embolus, or a chronic endarteritis. This condition is at times the sole diseased condition of the heart; at other times it is a part of a general sclerotic change that also involves the valves and the endocardium. These indurative changes are oftenest found in the wall of the left ventricle or in the papillary muscles.

Chronic indurative myocarditis is to be suspected where we find evidences of a weak heart with no valvular lesion, no acute infectious disease, no anæmia, no evidence of fatty change to account for the altered action. The complaint of the patient is practically that made in cases of non-compensated valvular disease. There is shortness of breath, a feeling of oppression in the chest. At times a dull pain, palpitation, and, as the circulation grows weaker and weaker, evidences of venous stasis in more distant organs manifest themselves, *e. g.*, œdema of the extremities, digestive disorders, diminished urine, and albuminuria. An examination of the heart in these cases reveals the fact that the heart is usually, though not always, increased in size, that the tones are pure, but the sounds feeble. The pulse at the wrist is feeble and very frequently irregular. It is common, in fact, to look upon irregularity of the heart without valvular lesions as an evidence of myocarditis.[1] Yet it is to be remembered that the same irregularity, and associated with practically the same symptoms, is to be found in cases of fatty heart. In some cases of myocarditis there are severe attacks of angina, and occasionally epileptiform seizures.

FATTY HEART.

Fatty heart may present exactly the same train of symptoms as does the myocarditic heart—shortness of breath, palpitation, irregular pulse, sense of oppression, evidences of venous stasis, with occasional attacks of vertigo, faintness, or of angina.

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[1] "The most striking symptom is the irregularity in the strength and rhythm of the heart tones. At times the first sound is loud, even valvular; at times it is but very slightly accentuated, or very much muffled. Now one hears a succession of tones rapidly following one another; then there ensues a pause, or several very slow beats follow, and so there is a continual variation of the rapid with the slow sounds, the weak with the strong, so that in certain cases for a long period it may be very difficult to determine whether there is a murmur or not. Fraentzel: *Vorlesungen über die Krankheiten des Herzens*, vol. iii, p. 10.

Two conditions are recognized pathologically under the heading of fatty heart. One is a true degeneration, such as occurs in the aged, in the anæmic, in cachexias, fevers, in phosphorus poisoning, in disease of the coronary arteries, and in many hypertrophied hearts; the other a fatty overgrowth or obesity of the heart, where the sub-pericardial fat is in excess, and may extend between the muscular bundles, even down to the endocardium. Many of the symptoms in these latter cases may be due to an accompanying pressure atrophy of the muscular fibres, as well as to a mechanical interference with the heart's action. In cases of fatty heart, as also in cases of myocarditis, sudden death occasionally occurs.

CORONARY DISEASE.

Coronary disease, under which term must be included embolism and thrombosis of the coronary arteries, as well as endarteritis obliterans, may be productive of many symptoms spoken of under the head of myocarditis and fatty heart. In fact, this condition is the most frequent cause of the indurative or fibrous myocarditis. With coronary disease attacks of faintness, vertigo, tachycardia, angina, even sudden death, are not infrequent. It is commonest in connection with cases where there are general arterio-sclerotic changes, and particularly where the aorta and the aortic valves are sclerotic.

IDIOPATHIC HYPERTROPHY AND DILATATION OF THE HEART.

Where the heart is enlarged and no valvular lesion can be found to account for it, nor any disease of the kidney or of the general arterial system, as arterio-sclerosis, nor lung disease, as emphysema, the condition is known as primary idiopathic hypertrophy. The cause in these cases can oftentimes be found in excessive physical exertion, as in the case of laborers and of athletes; in overfeeding and overdrinking, as in the beer drinkers of Munich, or in some nervous irritation of the heart, causing it to be overworked, as perhaps in exophthalmic goitre. The disease is often unrecognized until compensation begins to fail; then the ordinary symptoms of failing compensation are manifest—the same shortness of breath, palpitation, as occur late in valvular diseases. Examination of the heart reveals that it is enlarged, but also that there is no valvular lesion to account for the hypertrophy. The diagnosis is therefore made by the exclusion of the common causes of hypertrophy, by the symptoms of failing compensation, the evidences of cardiac enlargement, and a knowledge of the existence of some such cause as overwork or overdrinking. A differential diagnosis between idiopathic hypertrophy, chronic myocarditis, and fatty heart is in some cases impossible.

NEUROSES OF THE HEART.

ANGINA PECTORIS.

Severe pain referred to the heart, shooting into the left arm, up the left side of the neck, and accompanied by great anxiety, often a sense of impending danger, and by more or less shock, is spoken of as angina pectoris. These attacks come on suddenly, may last from a few seconds to several minutes, and may be fatal. Among the causes assigned, are obstruction in the coronary arteries, perhaps due to spasm of the arterial wall, excessive smoking, diseases of the valves, particularly the aortic valves, myocarditis, and fatty degeneration. The typical cases cannot be mistaken for anything else, though in nervous and hysterical patients intercostal neuralgias, muscular or gastric pains, may be exaggerated and referred to the heart, and in this way mistaken for angina pectoris.

PALPITATION.

Palpitation of the heart, *i. e.*, where the patient feels the action of the heart, is oftentimes but a symptom of some organic disease, but in not a few instances the most careful examination in cases of palpitation fails to reveal any organic disease, so that we must look upon this form of nervous palpitation as a pure neurosis. In these cases the attacks of palpitation come on under the slightest excitement. The heart is irritable, sensitive to external excitation, or there is "a kind of hyperæsthesia of the patient to the movements of the heart."

The diagnosis presents little difficulty when, on careful examination, we find that there is no objective change in the heart. The condition is found oftenest in the neurotic, in the anæmic, in those who indulge to excess in smoking, or the drinking of tea and coffee.

TACHYCARDIA.

Tachycardia is a name given to an excessive frequency of the pulse coming on in paroxysms. In many cases the cause is unknown, though it is oftenest found in the obese, in the nervous, in the anæmic, or following the acute infectious diseases. Drinking, smoking, errors in diet have been thought to produce it. The attack usually occurs suddenly, is accompanied by restlessness and anxiety, and at times great dyspnœa. Increase in the heart's dulness during these attacks has been observed. The paroxysmal nature of the disease, the enormous increase in the rapidity of the heart's action, render the diagnosis easy. Cases of tachycardia should always be carefully investigated for other evidences of exophthalmic goitre. It is often one of the earliest symptoms of that disease.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Primary pericarditis is mostly the effect of traumatism. Secondary pericarditis, the common form, is most frequently met with as a complication of rheumatism. It is also found in the course of infectious diseases, as pneumonia, typhoid, measles, erysipelas, diphtheria; also as a part of the pyæmic process. Patients affected with Bright's disease, with scurvy, and other pupuric diseases, seem to be predisposed to pericardial inflammation. The extension of an inflammatory process from neighboring organs may cause the pericardium to become involved. In this way, the tubercular variety of pericarditis is oftenest produced, as well as the carcinomatous, though, in either the tubercular or the carcinomatous variety, metastasis may account for the involvement of the pericardium. More rarely, tubercle or carcinoma is primary in the pericardium.

Pathologically, there are recognized the following varieties: Fibrinous, serous or sero-fibrinous, where the exudate is a clear serum or serum mixed with fibrin, purulent, and hemorrhagic, as well as the tubercular and cancerous varieties just spoken of. Males are oftenest affected with the disease.

The physical signs of pericarditis on which the diagnosis chiefly rests are sometimes trifling, and vary much, according to whether or not a fluid exudate is present. In the fibrinous variety, when the roughened pericardial surfaces are rubbing the one upon the other, a friction sound gives evidence of the inflammation. This sound is best heard near the base of the heart, oftenest, perhaps, in the third and fourth interspaces. It is usually a to-and-fro sound, that is, with the systole and the diastole, or it may be broken up into three or four parts; that is to say, with each beat of the heart two or three broken sounds are heard. The sound seems to be near the ear. Pressure with the stethoscope, and change of the position, may cause the sound to become louder. By palpation a friction fremitus can occasionally be felt. The symptoms in this early stage of the disease may be very slight, or pain may call attention to the affection. There is usually a slight rise of temperature and increase in the rapidity of the pulse.

It is not always easy to differentiate between a pleural friction rub and a pericardial friction sound, but the pleural rub stops when breathing stops. Much more difficult, and oftentimes impossible, is it, to differentiate between a pericardial friction rub and a pleuro-pericardial friction rub. But the latter sound is oftenest heard where there is evidence in other places remote from the pericardium, of involvement of the pleura, as in a tubercular or pneumonic process. Hard breathing influences the sound, and particularly with expiration the pleuro-pericardial friction may disappear. Careful examination is also necessary to distinguish aortic valvular murmurs from a pericardial friction sound.

When there is an exudate of whatever character, other signs appear. Inspection will show a bulging of the interspaces, at times a pulsation or wave-like motion in the præcordial space. The apex-beat may not be seen. Percussion shows dulness, and this of a peculiar form where the amount of fluid is great. The normal heart's dulness appears increased in all directions, but particularly to the left and upward. It will also extend to the right of the sternum. The shape of this dulness is pyriform or trapezoidal, the base being downward. Dulness is frequently noted in the fifth right interspace, when there is much fluid in the pericardial sac. It may extend downward, even to the eighth rib. It is of the greatest diagnostic importance to note that the dulness in pericardial effusion reaches beyond the point where the apex-beat is felt. On palpation the impulse of the heart is found to be weak, oftentimes imperceptible. Fluctuation is in some instances made out. On auscultation the heart sounds are distant and muffled. They are at times scarcely audible. No friction sound is heard with a large effusion. That the heart is not really weak, though the sounds are not clearly heard, is proven by the fact that the pulse is frequently strong and of good quality. This is of importance in differentiating between a dilated heart and pericarditis with effusion. Irregularity of the pulse is not infrequent in pericardial effusion.

The symptoms of pericardial effusion are frequently utterly lacking. "I know of no affection which is more frequently overlooked during life than pericardial effusion. This occurs because its development takes place without symptoms." (Musser.) In a few instances mental symptoms, such as mild or even severe delirium, have been noted. In other cases pressure symptoms are pronounced. Among these must be mentioned dyspnœa, or even orthopnœa, difficulty in swallowing, an irritative cough, hoarseness due to pressure on the recurrent nerve, and alterations in the rhythm of the heart's action.

FIG. 37.

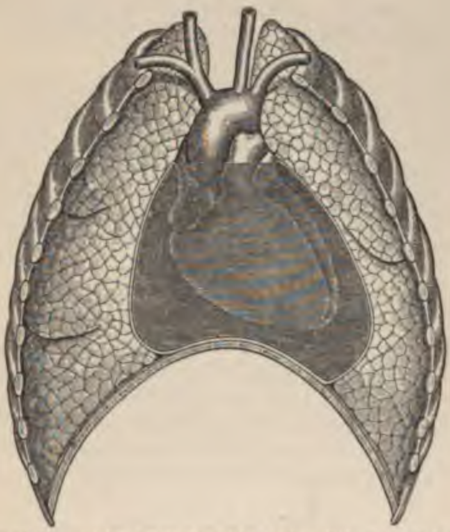


Illustration of the position of the heart in pericarditis, and of the distention ot the pericardium with fluid. The heart sounds are indistinct, except above the effusion; the impulse is feeble. The extent and shape of the percussion dulness may be judged of by the appearance of the distended sac. (DA COSTA.)

Differential Diagnosis. From *hypertrophy* of the heart, pericardial effusion is recognized by the lessened strength of the apex-beat, the muffled cardiac tones, the irregularity of the pulse, and by the fact that the dulness extends beyond the point where the apex-beat is seen and felt. From *dilatation* it is recognized by the peculiar outline of the dulness, which, in dilatation does not extend upward in so marked a degree; also by the absence of pressure symptoms and by the weak pulse of dilatation. From *pleurisy* with effusion it is recognized by the peculiar shape of the dull area, its location, the altered cardiac tones. With pleurisy the physical signs are usually as well marked behind as in front. Pericardial effusion rarely causes dulness below the eighth interspace in front, pleurisy a little lower.

Hydropericardium.

The physical signs of this condition are identical with those of pericardial effusion. It occurs in connection with disease of the heart, or with Bright's disease. In all cases of nephritis, hydropericardium should be carefully sought for. It is often associated with hydrothorax.

Hæmopericardium.

Rupture of bloodvessels or of the heart itself, wounds of the pericardium, or hemorrhage in the course of the hemorrhagic diseases may produce this condition. The physical signs are the same as those of hydropericardium, and the character of the fluid can only be surmised or determined by exploratory puncture.

Pneumopericardium.

This condition is very rare. It may be due to the entrance of air through a perforation from without, as from traumatism, or from within, as from rupture of the œsophagus, stomach, or some portion of the respiratory tract. A tympanitic note would be found over the heart, and when fluid accumulates, as it soon would from the result of suppurative pericarditis, there would be heard a metallic succussion sound.

Adherent Pericardium.

Adherent pericardium frequently produces no signs or symptoms during life, and is discovered accidentally post-mortem. In other cases the interference with the cardiac action is so great as to cause the symptoms of uncompensated valvular disease or of myocarditis. This is particularly true, where, in consequence of the pericardial inflammation, the myocardium has also become involved and the muscular fibres have undergone atrophy or degeneration. Here the clinical picture will be that of myocarditis.

Where there are external pericardial adhesions as well, the interference with the heart's action may be extreme. Among symptoms on which the greatest reliance can be placed is the systolic retraction of the chest wall, particularly the interspaces, most marked near the apex. Collapse of the cervical veins during diastole is also noted, as well as a small, feeble pulse during inspiration, the so-called *pulsus paradoxus*. Late in the course of this affection the physical signs are those of a dilated heart, perhaps with the development of murmurs and with general stasis of the venous system, characteristic of the late stages of dilatation. Too much importance should not, however, be attached to the retraction of the chest wall, or the collapse or undulation of the cervical veins, as these phenomena are also observed, at times, in other conditions.

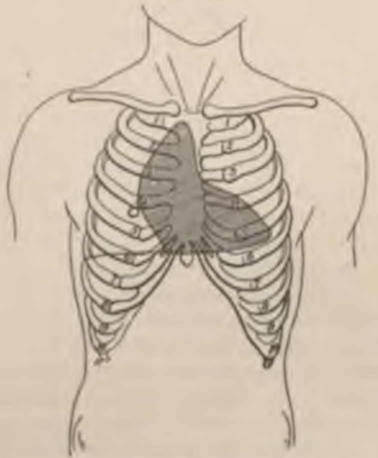
ANEURISM OF THE THORACIC AORTA.

Small aneurisms of the aorta may exist and cause no symptoms, or may cause symptoms not well enough defined, in the absence of physical signs, to enable one to make a diagnosis during life. Larger aneurisms are recognized by the physical signs and by the pressure symptoms. The physical signs and the symptoms will of course depend largely upon the location and size of the aneurism. Oftenest we find the ascending aorta affected, less frequently the arch or the descending aorta.

Inspection reveals an enlargement of the chest over the distended vessel. In aneurism of the ascending aorta, the bulging is to the right of the sternum in the first and second intercostal spaces. Pulsation may be seen over the protruding mass. In some cases, where the ribs and the sternum have become destroyed by pressure atrophy, we may see a distinct tumor-like mass, with pulsation more or less plainly marked. Palpation sometimes detects a systolic thrill and seldom fails to reveal a pulsation. Where the hard parts do not interfere with palpation we may detect an expansile pulsation, that is, a pulsation in all directions, not merely an up-and-down movement of the mass. This expansile pulsation is one of the surest indications of an aneurism. On percussion there is dulness over the mass. Auscultation may be negative, though we usually hear the transmitted heart sounds in every case. Frequently there is a distinct systolic murmur due to the vertiginous movements of the blood in the aneurismal sac. Often, also, a diastolic murmur which is probably due, in many cases, to the insufficiency of the aortic valves that is so frequently found with an aneurism.

The symptoms and signs in the parts remote from the aneurism are chiefly those of pressure. The heart may be pushed to one side or the other, according to the location of the dilatation. Compression of an artery will lead to weakening of the pulse in that artery. Thus, the two subclavians or the two radial arteries may, by comparison, be found to beat with unequal strength. So, also, there is frequently found a marked delay of the pulse in the arteries that arise below the aneurism. Compression of the veins will lead to venous stasis and marked œdema in the parts from which these veins spring. One arm may be œdematous, or the head and neck; or the thoracic veins may be markedly dilated. Compression of the lungs will lead to atelectasis; of the trachea and of the bronchi to the symptoms of tracheal and bronchial stenosis, inspiratory dyspnœa. One or the other recurrent laryngeal nerve may be pressed upon, and paralysis of the vocal cords produced, evidenced by hoarseness, difficulty in phonation, and immobility of the vocal cords. Pressure upon the branches of the brachial plexus or upon the intercostal nerves may cause severe neuralgic pains in the territories supplied by these nerve. Pain and difficulty on swallowing may result from pressure on the œsophagus.

FIG. 38.



Dulness in a case of aneurism of the ascending aorta. (BURY.)

The diagnosis of aneurism, as has been said, in some cases is impossible owing to the small size of the sac. In every case where there is distinct bulging of the chest wall, a pulsating tumor with a bruit, the diagnosis is easy. The differential diagnosis, however, between intrathoracic tumors, tumors of the bronchial glands, circumscribed empyemas and other abscesses, is not always easy. We are aided always in making a differential diagnosis by the presence of the swelling, its pulsation, particularly expansile pulsation, the presence of a murmur within the swelling, and also by the condition of the heart and the arteries. Aneurism oftenest occurs where there are other evidences of arterio-sclerosis. It is frequently found, therefore, in connection with sclerotic disease of the aortic valves. Often, too, there is a history of syphilis, gout, rheumatism, or lead poisoning.

The formation of parietal thrombi in the aneurismal sac may alter the signs from day to day. Thus, variations in the bruit, in the strength of the radial pulse, and even in the pulsation, are not uncommon, the thicker the thrombotic deposit, the less marked, as a rule, the bruit and the pulsation.

The caution against the use of the œsophageal bougie for purposes of diagnosis in suspected œsophageal obstruction, without first excluding a pressure stenosis from a thoracic aneurism, cannot be too often repeated.

ARTERIO-SCLEROSIS.

Arterio-sclerosis, a condition in which the walls of the vessels are thickened from chronic sclerotic changes, is to be regarded in most instances as a primary disease; in others, perhaps, as secondary to primary fibrous changes in the kidneys.

It is always important to recognize this condition, as it explains in many instances the changes that have taken place in the kidney, the heart, the brain, the liver. The blood vessels that are open to inspection and palpation are seen to be tortuous, often pulsating visibly, and are felt to possess thickened walls. The temporal vessel is one easy of observation. The radial artery shows in cases of arterio-sclerosis that its wall is thickened, and gives evidence of cardiac hypertrophy, by the full, tense character of the pulse, which is hard and unyielding. Compression of the artery between the wrist and the heart, so that the circulation is shut off, allows the artery below, with its thickened walls, to be felt as a hard, fibrous band. The radial artery with normal walls cannot in this way be palpated.

In many cases of arterio-sclerosis the symptoms are chiefly those of renal disease. The increased amount of urine, of low specific gravity, with traces of albumin, and a few hyaline and granular casts, shows that a chronic interstitial process is going on. The heart, too, in most cases, is hypertrophied, and sclerotic changes upon the valves, particularly the aortic, may give the physical signs of valvular disease. Fibrous myocarditis, or sclerotic changes in the coronary arteries, may cause cardiac weakness and arrhythmia, as already described under this heading. Sclerosis of the arteries of the brain may lead to thrombosis and the symptoms of cerebral softening. The lumen of the bloodvessel may, by the obliterating endarteritis, be completely occluded, or a thrombus may form within the bloodvessel. The effect of the occlusion of such a vessel depends, of course, upon the parts supplied by it. If it is in a peripheral vessel, as of the leg, gangrene of the part may follow; if in the brain, as already mentioned, the evidences of cerebral thrombosis Aneurismal dilatations are common in sclerotic vessels and the rupture of miliary aneurisms of cerebral vessels is the most frequent source of cerebral hemorrhage.

CONSTITUTIONAL DISEASES

(INCLUDING DISEASES OF THE BLOOD AND DUCTLESS GLANDS).

CLINICAL EXAMINATION OF THE BLOOD.

For clinical purposes the blood is to be examined as follows:

1. Naked-eye appearances.
2. Microscopic examination of the red corpuscles. (*a*) Number; (*b*) size; (*c*) shape; (*d*) color; (*e*) nuclei and contents.
3. White corpuscles. (*a*) Their number and relation to the number of red corpuscles; (*b*) size; (*c*) shape; (*d*) nuclei; (*e*) granular matter and its reaction to stains.
4. Hæmoglobin and its relation to the number of red corpuscles.
5. Specific gravity.
6. Micro-organisms.
7. Chemical analysis, reaction, percentage of CO_2, etc.
8. Spectroscopic examination, which is necessary in certain cases of poisoning.

Manner of Obtaining Blood.

Blood for the purpose of examination can best be obtained from the finger or lobe of the ear. The part should be thoroughly cleansed both with water and alcohol, and when dry, a puncture is made with a sterilized needle or a small lance, deep enough so that the blood flows without the aid of pressure. It is better to take the second drop than the first.

1. Macroscopic Examination

merely shows the color of the blood and its rapidity of flow. The trained eye can form some estimate, by the shades of color and the rapidity of flow, as to the number of red corpuscles or percentage of hæmoglobin, and the total amount of blood in the body.

2. Microscopic Examination of the Fresh Specimen.

A drop of blood is placed upon the clean slide and carefully covered with a cover-glass; to prevent too rapid drying of the specimen, vaseline can be placed about the edges of the cover. The red corpuscles are examined first as to **form**. Normally, the red corpuscle is biscuit-shaped, with a central depression. Often the corpuscles appear in rouleaux; in severe anæmias the rouleaux may not be present. **Poikilocytosis** is a term used to designate the variations from the rounded contour of the corpuscles seen in such conditions as pernicious anæmia. The corpuscles may be kidney-shaped, pear-shaped, sickle-shaped, or with buds and protuberances here and there, such as are seen in white corpuscles during amœboid movements. The red corpuscles in conditions of disease may vary greatly in **size**. The smaller corpuscles are here spoken of as **microcytes**; the larger ones, often ovoid, as **megalocytes** or **macrocytes**. Such variations may be found in pernicious anæmia. The **color** of the corpuscles, where hæmoglobin is deficient, as in chlorosis, is pale, and may be recognized as such by the trained eye. Normally, no nucleus is visible in the red blood globule. In some pathological states nuclei can be seen, best, of course, in the stained specimen. Thus, in progressive pernicious anæmia and late in leukæmia, there may be seen small nucleated red corpuscles, to which the name **normoblasts** is given, or larger nucleated red corpuscles known as **gigantoblasts**. Within the red blood-corpuscles parasites are sometimes seen in the stained or in the unstained specimens. Thus, the malarial plasmodium may be visible; foreign bodies, *e. g.*, pigment, can also be seen.

The **number** of red blood-corpuscles in man is, in the condition of health, about five millions to the cubic millimetre; in woman, between four and five millions. In many pathological conditions the number of red blood-corpuscles is markedly diminished; it is, therefore, of great importance to be able to estimate their number. Several instruments have been devised for this purpose, among the best of which are Gowers's hæmocytometer and the Thoma-Zeiss blood counting apparatus.

This latter instrument consists of a glass capillary tube, with a bulbar enlargement which contains a small glass ball. This tube serves for the purpose of sucking up the blood and diluting it. The other part of the instrument is a counting chamber. The blood is sucked in the graduated tube to the mark 5 or 1, then the tip of the tube is wiped off and enough 3 per cent. salt solution is sucked up to cause the fluid to rise to the point 101.[1] The fluid is mixed in the bulbar enlarge-

FIG. 39.

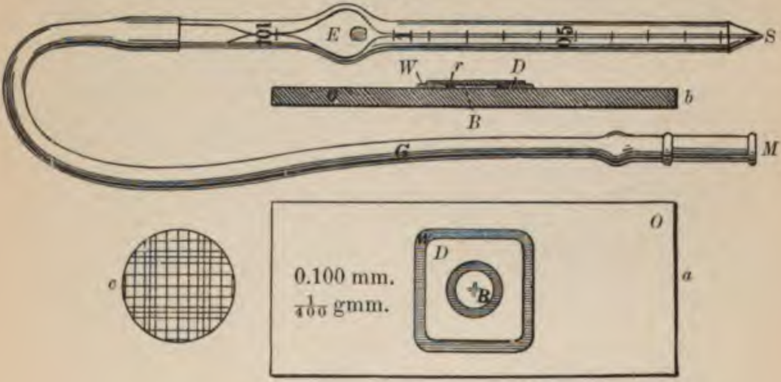
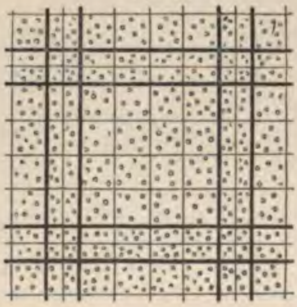


FIG. 40.



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[1] Other mixing solutions may be employed. A 2½ per cent. solution of potassium bichromate is commonly employed in von Jaksch's clinic at Prague.

By the use of Toison's solution, which stains the white corpuscles, there is less confusion in determining under the microscope which are red and which white corpuscles. The formula for Toison's solution is as follows:

Methyl violet	.025
Neutral glycerin	30.
Distilled water	80.

To this add:

Chloride of sodium	1.
Sulphate of sodium	8.
Distilled water	80.

Filter.

The leucocytes stain in about ten minutes.

ment by vigorous shaking, then the salt solution in the capillary tube is blown out and a drop of the solution of blood is placed in the counting chamber. This chamber is exactly one-tenth of a millimetre deep, and its floor is divided into microscopic squares. The space over each square, when covered with a cover-glass, contains $\frac{1}{4000}$ of a cubic millimetre. The cover glass is carefully placed over the blood, so that no air-bubbles are present. The instrument is allowed to remain quiet for a few minutes, so that the corpuscles shall have time to settle, and then the number of blood corpuscles in sixteen squares is counted, and the average number in each square determined. This number multiplied by 800,000 gives the number of red blood-corpuscles in a cubic millimetre of blood. If the blood was drawn up to the mark 1, then the number in each square must be multiplied by 400,000. In order to include in the counting of the corpuscles within each square, and to avoid counting twice, those that are upon the lines, it is well to adopt the rule to count as in a square, those touching the upper and left borders of each square, not counting those touching the right and lower borders.

3. White Corpuscles.

The number of white corpuscles can often, at the first glance through the microscope, be seen to be increased above the normal. With an ordinary objective (Leitz No. 7) rarely more than three or four white corpuscles are seen in one field. In leukæmia, however, one hundred or more may, at times, be seen in a single field. Normally, the relation between the white and red corpuscles is as 1 to 350–600. The white corpuscles can be counted by the Thoma-Zeiss apparatus in the same manner as the red corpuscles. The count is aided by coloring the salt solution with a little methyl-blue. This stains the white corpuscles and leaves the red corpuscles unchanged. With the Thoma-Zeiss instrument there is also a special mixing pipette for white corpuscles. By diluting the blood with a $\frac{1}{3}$ per cent. solution of acetic acid, the red globules are destroyed, and the white alone remaining in the field, are readily counted. Normally, there are 5000 to 8000 white corpuscles in a cubic millimetre. A moderate increase in the number of white corpuscles (this increase solely in the polynuclear neutrophiles) is **leucocytosis**. Leucocytosis is physiological after meals, and is found in many diseased conditions, as, for example, pneumonia, carcinoma, etc. Over 50,000 white corpuscles to the cubic millimetre may usually be safely asserted to mean leukæmia; yet von Limbeck[1] reports a case of leucocytosis accompanying carcinoma of the kidney with metastatic nodules in the suprarenal capsule and the retro-peritoneal and mediastinal lymph glands, in which over 80,000 white blood-corpuscles were found in a cubic millimetre.

The mobility of the white blood-corpuscle is not of much clinical value, but it is lessened in leukæmia. The polynuclear leucocytes alone are motile. The size, shape, nuclei, and granular matter of the leucocytes are best observed in stained specimens. For staining specimens of blood, the following directions should be followed:

a. Cleanse the slides, covers, and the part from which the blood is to be obtained, with water, alcohol, and ether.

b. Prick the finger or lobe of the ear so that blood flows freely, and obtain as thin a coating of blood as possible upon the cover-glass, by quickly sliding one cover over the other, the covers being held by forceps.

c. Dry in air.

d. Fix. This can be done by immersing the cover in equal parts of absolute alcohol and ether for a few hours. For quick work and not permanent specimens, immersion for ten minutes will suffice. The specimen may be fixed by heating for two hours at a temperature of 120° C. This can be readily done by heating a copper bar by means of an alcohol lamp placed beneath it, and placing the covers upon the copper bar, just within the point at which a drop of water placed upon the copper bar boils.

e. Stain. For staining white blood corpuscles both basic and acid dyes, as methyl-blue and eosin, are best employed. Many formulæ for stains are given. A good one, easy of application and reliable, is as follows: Eosin, 0.5; alcohol, 100.0. The cover-glass is stained for five minutes in this solution, which has been diluted one-half with water. Wash with water, air dry, and stain for forty seconds in a saturated aqueous solution of methylene blue, which should be diluted one-half with water before using. This stains the red corpuscles red, the nuclei blue, and any parasites blue.

Ehrlich has shown that the granular matter in the white blood corpuscles stains differently. He names these granules according to the Greek letters of the alphabet—α, β, γ, δ, ε. Those of clinical importance in blood study are the Alpha granules—the acid-staining, oxyphilic, or eosinophilic granules—and the Epsilon, or neutrophilic, those staining in a combination of the acid and basic dyes. The basophilic granules, or those staining with basic dyes, are rarely found in the blood, though often in inflammatory regions. Red corpuscles take an acid stain; nuclei, a basic stain. A triple stain containing methylene green, acid fuchsin, and orange G., is often employed, and with this stain the red corpuscles show a reddish-orange tint, all nuclei are green, the eosinophilous granules are red, the neutrophilic granules of a lilac color.

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[1] Grundriss einer Klinischen Pathologie des Blutes, p. 154.

Varieties of White Corpuscles.

Ehrlich recognizes five varieties of white blood corpuscles:

a. **Small lymphocytes,** about the size of a red blood-corpuscle, possessing a single, large, intensely staining nucleus, surrounded by a very narrow rim of protoplasm.

b. **Large lymphocytes,** about twice the size of a red corpuscle, with a protoplasmic mass fairly well defined. Both varieties of lymphocytes are supposed to have their origin in the lymph glands.

c. The so-called **mononuclear transitional forms.** The nucleus is more or less curved upon itself and the body of the corpuscle shows the first signs of the so-called neutrophilic granulations.

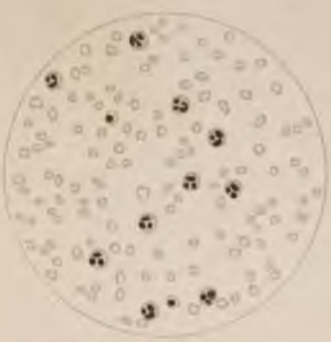
d. **Polynuclear leucocytes.** These are larger than the red blood-corpuscle, possess a nucleus of irregular shape (S-shape, kidney-shape), or sometimes two or three nuclei. The granular matter takes only the neutral stain, that is, a combined basic and acid stain.

e. **The eosinophile.** The eosinophile is large, with a plainly marked, deeply staining nucleus, often of an irregular shape, and with large refractile granulations that take the acid dye. These granulations in their glistening appearance resemble somewhat small fat globules.

In normal blood these various corpuscles are found in about the following proportions: Small lymphocytes, 20 per cent.; mononuclear lymphocytes and transitional forms, 5 per cent.; polynuclear neutrophiles, 70 per cent.; eosinophiles, 2 to 5 per cent. In abnormal conditions of the blood the white corpuscles may be found in varying proportions, and in some cases are accompanied by a different variety of white blood-corpuscle known as *myelocyte.* The myelocyte is a large, mononuclear leucocyte with fine neutrophilic granulations. It is found chiefly in certain varieties of leukæmia. The practical

PLATE II.

Fig. 1.

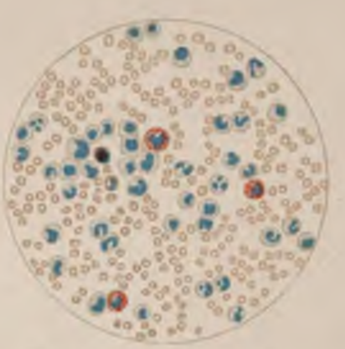


Severe Anæmia with Leucocytosis.

Dry preparation. Fixed with picric acid. Stained with hæmatoxlin Böhmer, x 300.

Red corpuscles few, almost colorless, varying in size, show poikilocytosis; two nucleated reds (normoblasts). The increase in the white cells seen to be in the polynuclear elements. (Rieder's "*Atlas der Klinischen Mikroskopie des Blutes.*")

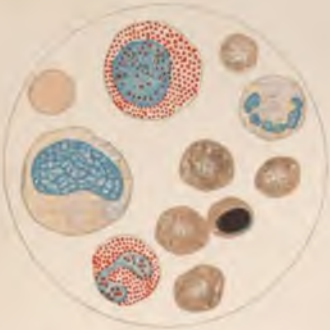
Fig. 2.



Splenic-myelogenic Leukæmia.

Eosin-hæmatoxylin, x 300. Red corpuscles rosy-red, of nearly uniform size, round. To the left a normoblast with eccentrically placed nucleus. Many large mononuclear leucocytes (myelocytes) and three eosinophiles seen. (Rieder).

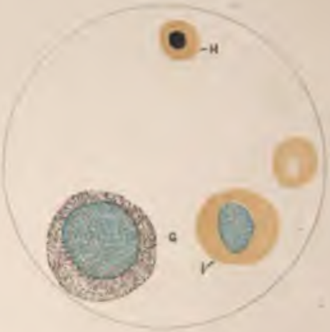
Fig. 3.



Splenic-Myelogenic Leukæmia.

Same case. Eosin-hæmatoxylin, x 1100. One normoblast, one polynuclear leucocyte, one myelocyte, two eosinophiles. The neutrophilic granules of the polynuclear leucocyte and of the myelocyte do not show with this stain. The large mononuclear eosinophile above is believed to be also a myelocyte (Markzelle), the smaller one below, an eosinophile such as can be found in normal blood. *(Rieder)*.

Fig. 4.



Myelocyte, normoblast, megaloblast.

Triple stain. G, myelocyte showing neutrophilic granules; H, normoblast, both from a case of splenic myelogenic leukæmia; I, large nucleated red corpuscle (megaloblast) from a case of pernicious anæmia. (Osler.)

value of Ehrlich's method of staining blood will be shown in considering the diagnosis of the various diseases of that tissue.[1]

(For the varieties of red and white blood-corpuscles, see Plates I. and II.)

4. Percentage of Hæmoglobin.

The percentage of hæmoglobin is best determined by means of either Gowers's or Fleischl's instrument. Fleischl's hæmometer, which is, perhaps, the best, consists of a red glass wedge mounted beneath a platform, like that of a microscope, with a circular opening in the centre. Upon this opening light from a gas or oil lamp, or a candle (not from the sun), is thrown by a plaster-of-Paris plate. Above the wedge and over the circular opening in the platform is a metallic tube, closed at the bottom with a glass plate and divided by a vertical metallic partition, so that one-half of the tube receives light through the red glass wedge, the other directly from the light reflector. When in use the first compartment is filled with water, the other with water mixed with a known quantity of blood. This quantity is that contained in the pipette accompanying the instrument, of such size that when healthy blood is used the resulting mixture corresponds in color to that derived from the part of the red glass wedge, marked 100. To use the instrument a capillary tube is filled with blood (care being taken that none is upon the outside of the tube) and placed in the blood chamber, which contains a little water with which the blood is then intimately mixed. Both compartments are filled with water, the blood in the blood chamber being thoroughly

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[1] For general work and as an all-around stain the eosin-methyl-blue stain is as good as any. Various modifications of this are employed. By this method there is an excellent stain given to all nuclei, to the malarial plasmodium, and to the eosinophilous granules.

Hæmotoxylin and eosin are also employed for this same purpose.

If the neutrophilic granules are to be brought out, the Biondi-Ehrlich triple stain or some modification is employed. A Biondi-Ehrlich powder is on the market. For staining, this can be used, making a saturated solution by dissolving in:

Water	6
Alcohol	1
Glycerin	0.5

If to this there is added a little additional acid fuchsin better staining effects are obtained.

As an equivalent of the Biondi-Ehrlich the following has been advised:

Saturated aqueous solution orange (G)	55.
Saturated aqueous solution acid fuchsin	50.
Distilled water	100.
Alcohol	50.

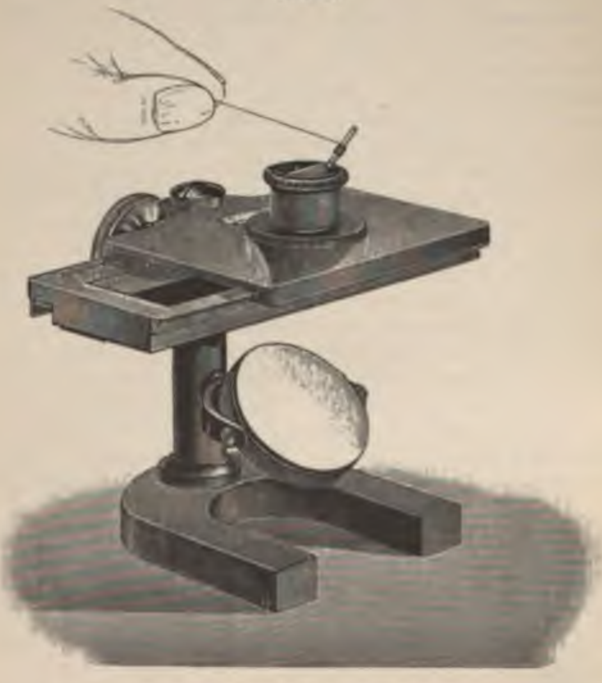
To this add:

Saturated aqueous solution of methyl-green	65.
Distilled water	50.
Alcohol	12.

Let stand for ten to twenty days before using.

mixed with the water, then the red glass wedge is moved until the two fluids show an equal intensity of color. The number indicated on the scale is then read off, the number representing the percentage of the normal proportion of hæmoglobin. It is rare to find, even in persons supposedly healthy, 100 per cent. hæmoglobin with the Fleischl instrument. And even with care there is always the possibility of there being a small percentage of error.

FIG. 41.



Von Fleischl's Hæmometer.

5. Specific Gravity.

Clinically, the specific gravity of the blood is of trifling importance as it varies with the number of red blood-corpuscles and the amount of hæmoglobin. The readiest method of determining the specific gravity is that of Roy. Several mixtures of glycerin and water, with specific gravity varying from 1035 to 1065, are taken, and into each of these is placed a drop of blood. The specific gravity of the mixture in which the blood drop swims is that which represents the specific gravity of the blood. Hammerschlag employs practically the same method, using a mixture of chloroform and benzol. This method has the advantage of employing but one solution, the specific gravity of which is varied by the addition of chloroform or benzol, as the case may be, until a solution is secured in which the blood drop swims.

6. Micro-organisms.

Micro-organisms in the blood can be detected by the methods of staining of Ehrlich, Weigert, Gram, and others, described in works upon bacteriology. The malarial plasmodium is excellently shown, stained blue, by the eosin and methyl-blue method previously described.

7 and 8. Chemical and Spectroscopic Examination.

These examinations are of such limited practical value, and of such complicated detail, that no description of them is included in this work.

LEUCOCYTOSIS.

By leucocytosis is meant an increase in the number of polynuclear leucocytes. This condition is physiological in infants during the first few days after birth, in pregnancy, in many well-nourished, robust, healthy individuals, and after meals. Certain drugs, as tonics and some of the ethereal oils, are said to produce leucocytosis.

Pathologically, leucocytosis is found in febrile diseases accompanied by exudation. It is found to a marked degree in pneumonia; it is also found in pleurisy, peritonitis, suppurative meningitis, erysipelas. As a rule, the higher the fever in these diseases, the larger the number of leucocytes. It may be of aid in a differential diagnosis between different varieties of meningitis. Thus in purulent meningitis leucocytosis is pronounced. In tubercular meningitis there is no leucocytosis. In typhoid fever, malaria, tuberular troubles, measles, scarlatina, there is usually no increase in the leucocytes. A moderate degree of leucocytosis would be an increase of the leucocytes to 10,000 to the cubic millimetre. When fever disappears, the increased number of leucocytes likewise disappears, even though the exudate may remain. In pneumonia, just before the fever falls by crisis, there is lessened leucocytosis. Thus, in one day there will be a diminution of leucocytes from 20,000 to 10,000. Where there is fever and emaciation, as, for example, in typhoid fever, there may be a decrease in the number of leucocytes, so that instead of the normal number, from 5000 to 10,000, there may be but 2000 or 3000. The blood in cases of malignant tumors contains an increased number of leucocytes, irrespective of the location of the tumor, though the leucocytosis is most marked where the new growth involves the lymph glands. In examining for leucocytosis, it is to be remembered that the blood should not be looked at until several hours have elapsed since the last meal, since, following the meal, there is normally a digestion leucocytosis. Only by staining the blood can definite statements be made as to the existence of leucocytosis.

LEUKÆMIA.

There are three varieties of leukæmia, viz.: the splenic, the lymphatic, and the myelogenous. These varieties are oftener found in combination than separately; in fact, pure myelogenous and pure lymphatic leukæmia are extremely rare.

Clinically, leukæmia begins insidiously and with symptoms that differ in no respect from those of an ordinary anæmia. Malaria, syphilis, and particularly traumatism in the splenic region seem at times to be causal factors. There is complaint of weakness, languor, perhaps derangement of appetite, dizziness, tinnitus aurium, faintness, and palpitation. These symptoms, it will be noted, are such as might occur in any case of anæmia or chlorosis. Hemorrhages, notably epistaxis, may occur early in the course of the disease, or may be one of the later manifestations. The patient may consult a physician because of some of the symptoms mentioned, or may not think the illness of serious import until he detects an enlargement of the abdomen. This enlargement may be discovered accidentally, or pain may call atttention to the region of the spleen where the tumor can be felt. As the disease progresses, anæmia becomes more marked, and œdema of the dependent portions of the body may appear. Not infrequently there are nausea, vomiting, and diarrhœa. Priapism has been at times observed. A slight degree of fever, 99½ to 103°, is common. The pulse is soft, compressible, and increased in rapidity. The spleen is found upon examination to be enlarged, firm, reaching perhaps beyond the median line and extending at times even to the symphysis pubis. The surface is smooth; the splenic notch is usually detected; there may be tenderness upon pressure; there is dulness upon percussion.

The diagnosis can only be made positively by means of an examination of the blood. Where the number of white blood-corpuscles is over 50,000, it may be safely said that leukæmia exists, though in some cases, as in the case of von Limbeck just quoted, this number has been surpassed in cases of leucocytosis. The blood in leukæmia shows a diminution, rarely extreme, in the number of red corpuscles. The percentage of hæmoglobin is likewise diminished.

An examination of the white blood-corpuscles enables us to make a diagnosis not alone of leukæmia, but, if the specimen be stained, of the variety of the leukæmia. In lymphatic leukæmia the increase is solely in the small mononuclear lymphocytes.

In the *splenic-myelogenic* variety the striking change is in the presence of the abnormal *myelocytes*. These are large mononuclear leucocytes with the protoplasm filled with fine neutrophilic granules. There is little if any increase in the lymphocytes or in the polynuclear neutrophiles. The eosinophiles, though absolutely increased, are not, usually, relatively so, though a relative increase is occasionally noted. Nucleated red blood-corpuscles are frequently found. Poikilocytes, microcytes, and macrocytes may abound. It is to be remembered that many cases of leukæmia are combinations of the various forms, so that the blood may present in a single specimen, appearances characteristic of each of the different varieties of leukæmia.

Lymphatic leukæmia is rare, and is a more quickly fatal form than the splenic variety. The cervical, inguinal, and axillary glands may all be enlarged. The glands are usually soft, freely movable, no adhesions binding them to each other or to the surrounding tissues. The lymph follicles of the tongue and pharynx may be enlarged.

The *myelogenous form* in combination with the other varieties is probably commoner than is generally supposed. Pure myelogenous leukæmia is, however, very rare. Occasionally tender points over the bones may lead us to suspect the existence of disease of the bone marrow.

Differential Diagnosis. From **leucocytosis,** leukæmia is differentiated by the fact that in leucocytosis there is a moderate increase in the number of leucocytes, and this wholly in the polynuclear neutrophiles.

In the same way the examination of the blood in *Hodgkin's disease* accompanied by leucocytosis, enables us to make the differential diagnosis. It is to be remembered, however, that some cases of Hodgkin's disease seem to undergo a transformation into the lymphatic variety of leukæmia.

Malignant growths with glandular enlargement and anæmia, may simulate leukæmia. Here, again, the examination of the blood will reveal simply leucocytosis.

Anæmias are recognized by the greater decrease in the number of red corpuscles and a lack of increase in the white corpuscles.

The following table will show at a glance the percentage of the various varieties of colorless corpuscles in the blood of health, of leucocytosis, of splenic-myelogenic leukæmia, and of lymphatic leukæmia. The figures are, of course, only approximately correct, and are true for typical cases:

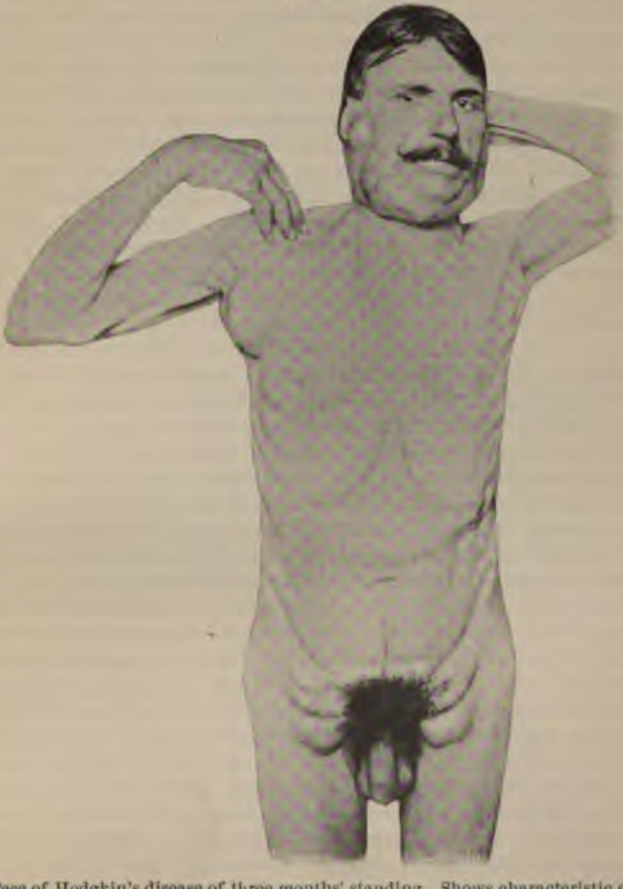
	Normal blood.	Leucocytosis.	Splenic-myelogenic leukæmia.	Lymphatic leukæmia. (Thayer, Shattuck's case.)
Small lymphocytes . .	15-30	4	1	97.9
Large mononuclear lymphocytes and transitional forms .	5 ±	6 ±	6 ±	0.4
Polynuclear neutrophiles	75	89	65	1.4
Eosinophiles . . .	3-5	1	5	0.1
Myelocytes . . .	0	0	23	0.0

PSEUDO-LEUKÆMIA, OR HODGKIN'S DISEASE.

Pseudo-leukæmia, or Hodgkin's disease, presents practically the same anatomical peculiarities as does leukæmia, save that in the blood there is not the same increase in the white blood-corpuscles.

The following varieties may be taken as types of the disease: 1. The form with an enlarged spleen, with no apparent involvement of lymph glands or bone marrow, sometimes spoken of as **splenic anæmia**. 2. The form in which the lymph glands are enlarged, **lymphatic anæmia**. 3. The form in which both spleen and lymphatic glands are enlarged, the typical **Hodgkin's disease.** The dividing line between this, the ordinarily accepted form of pseudo-leukæmia and certain malignant growths of the lymphatic glands, as sarcoma and lympho-sarcoma, is not clearly shown.

FIG. 42.



Case of Hodgkin's disease of three months' standing. Shows characteristic enlargement of palpable glands, including the cubital. Dulness over anterior mediastinal space due to enlarged glands. Deep palpation reveals enlarged abdominal glands. Spleen as yet not enlarged.

The symptoms of Hodgkin's disease are practically the same as those of leukæmia. It attacks chiefly young males. Syphilis, malaria, chronic skin diseases are assigned as causes. Usually the first symptom to attract attention is the enlargement of the submaxillary and cervical glands; later the axillary and inguinal glands are enlarged, as well as the lymph glands not visible to the naked eye. Dulness over the anterior mediastinal space may be marked because of the glandular masses beneath. The enlarged glands are usually discrete, soft, quite freely movable, rarely undergoing suppuration or caseation. They may attain a considerable size, even that of a fœtal head. The inconvenience produced by these enlargements is sometimes extreme. The neck may be encircled by a wide bulging collar of enlarged glands. Pressure symptoms are not unusual. Thus, pressure upon the œsophagus may produce dysphagia, upon the trachea, or a bronchus, interference with respiration, upon the laryngeal or pneumogastric nerves, interference with phonation or with the action of the heart. The glands are not usually painful, unless they make pressure upon the adjacent nerves. The spleen is often moderately enlarged. Tenderness over the bones points suspiciously to the involvement of the bone marrow.

The general symptoms are those of anæmia. There is failure in strength, more or less emaciation, and often derangement of the stomach and of the intestines, diarrhœa being not infrequent. The pulse is usually small, and frequently there is complaint of dyspnœa on exertion; a tendency to hemorrhages is common, especially epistaxis and metrorrhagia. Petechial spots are not infrequently seen on the lower extremities, or even over the entire body. Anæmic murmurs can be heard over the base of the heart. As the anæmia becomes more profound, dropsical symptoms may be manifested, thus hydrothorax, abdominal ascites, or even a universal œdema may be noted. The skin may be somewhat erythematous, and there seems to be a tendency to furunculosis. Rarely, cutaneous lymphomata have been observed. Fever appears in this disease as in the severe anæmias, and in some cases has been of a peculiar intermittent type, the intermissions and exacerbations each lasting for a period of several days. To this peculiar form of fever the name **chronic relapsing fever** has been applied. Pain is not infrequently complained of near the region of the spleen, or in the neighborhood of the larger swellings. Neuralgic pains in parts at a distance from the swellings are often noticed.

The examination of the blood shows a moderate diminution in the number of red blood-corpuscles, a corresponding diminution in the hæmoglobin. There is usually more or less leucocytosis,[1] and in some cases there develops a marked increase in the small lymphocytes, giving the blood the exact characteristics of lymphatic leukæmia.

The disease with which pseudo-leukæmia will be most readily confused is **tubercular adenitis.** But in tubercular adenitis the glands are usually slower in enlarging and extending; there is a greater tendency toward periadenitis and adhesion of the glands to one another, uniting them into firm bunches; the glands oftener suppurate or caseate; oftener, too, tubercular adenitis is unilateral, seldom as universal as in Hodgkin's disease; often tubercular foci in the lungs, kidney, bladder, and joints may be found. In doubtful cases a gland may be removed for the purpose of diagnosis.

ANÆMIA.

By anæmia is meant a condition of the blood in which there is a diminution in the volume of the blood, or in its corpuscles (oligocythæmia), or in its hæmoglobin (oligochromæmia). The anæmias are either primary or secondary. By primary anæmia is meant that form of the disease in which no other organs or tissues than the blood or the hæmatopoietic organs are affected. In secondary anæmia some other tissue than the blood is primarily in a pathological state. Of the primary anæmias there may be made three groups, viz.: Simple or constitutional anæmia, chlorosis, and progressive pernicious anæmia. The dividing lines between these three varieties are by no means clearly drawn, either pathologically or clinically.

The simple, constitutional anæmia is found oftentimes with no assignable cause. The patient appears somewhat pale, is perhaps slightly languid, suffers from a little palpitation, and dyspnœa. This condition is not incompatible with an active and enjoyable life. At times it seems to be congenital; at other times due to improper hygiene. Virchow has asserted that in some cases the arterial system is but imperfectly developed.

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[1] In a case of Hodgkin's disease under my observation in the Cook County Hospital in 1892, the relation of the white to the red corpuscles was as 1 to 47, but the increase was solely in the polynuclear elements, 90 per cent. of all leucocytes being polynuclear neutrophiles. Thus, a ready diagnosis between this condition and true leukæmia was made by the examination of the stained blood preparation.

CHLOROSIS.

Chlorosis is a temporary anæmia occurring oftenest in girls at about the age of puberty. While there is usually an assignable cause in poor hygienic surroundings, overwork, or a neurotic temperament, the disease is found at times where no cause can be discovered.

The **blood** in these patients is found to have nearly the normal number of red blood-corpuscles. Very frequently more than 4,000,000 are found in each cubic millimetre. The hæmoglobin, on the contrary, is greatly diminished, frequently being as low as 50, 40, or 30 per cent. of the normal. This disproportion between the number of red blood-corpuscles and the percentage of hæmoglobin is not found so strikingly in any other disease. Poikilocytosis may be marked in severe cases. Microcytes and macrocytes are frequently seen. There may be slight leucocytosis. There is a noticeable pallor of the skin and of the mucous membranes. The ears may be of almost waxy transparency. The adipose tissue is well preserved.

The subjective symptoms can be readily understood, if we remember that the blood, poorly loaded with oxygen, can but improperly nourish all the tissues and organs of the body. Thus the functions of all organs are but imperfectly performed. The **muscles** lack tone and strength, and the patient feels weak and languid. The **nervous system** is disturbed; there are mental weakness, incapacity for prolonged study, a tendency to drowsiness. Cerebral disturbances, such as tinnitus aurium, spots before the eyes, dizziness, are complained of. Neuralgic pains may be present in any of the peripheral nerves. Fainting spells, due to cerebral anæmia, are not uncommon. The **circulatory organs** functionate improperly; the pulse is rapid; the heart is extremely irritable, slight excitement or the least exertion causing it to beat with great rapidity. Anæmic murmurs are frequently heard, especially at the base of the heart. A venous hum is also heard over the veins of the neck. Amenorrhœa is occasionally observed. The **digestive tract** shares in this functional depression; the appetite is usually poor or extremely capricious, patients evincing a desire to eat indigestible or strange articles, such as chalk, slate-pencils, pickles, etc. Gastralgic attacks, aside from those induced by ulcer of the stomach, which may complicate the chlorosis, are frequent. The intestines, from lack of the normal secretion and because of deficient peristalsis, are constipated. The urine is usually light-colored, of high specific gravity, showing that it contains a large amount of solids. It is rarely albuminous. Polyuria is not infrequently noted. A slight elevation of temperature is occasionally observed. In some of the severer cases femoral thrombosis, or even sinus thrombosis, has been noticed.

Chlorosis is usually readily recognized, the mistake most often made, being that of regarding a secondary anæmia as chlorosis. A disease should never be pronounced chlorosis until all the organs of the body have been subjected to a careful examination and found to be healthy. The blood should be examined in all doubtful cases, and readily shows by the disproportionate decrease in the hæmoglobin the existence of chlorosis. The disease which is most frequently taken for chlorosis is incipient phthisis. The palpitation and murmurs at the heart may, unless the physician is careful, lead him to diagnose valvular disease, and if œdema be present, with some albuminaria, as occurs in the severer cases, Bright's disease may be wrongly suspected.

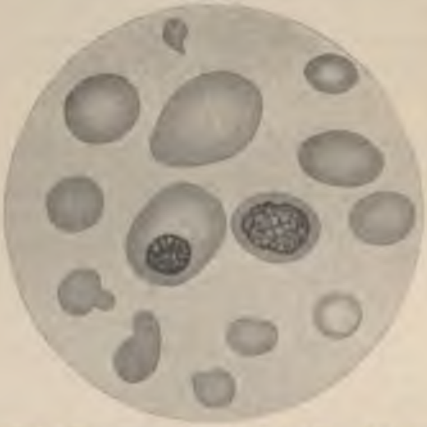
PROGRESSIVE PERNICIOUS ANÆMIA.

Careful clinical and post-mortem examinations have lessened considerably the number of cases of progressive pernicious anæmia. They have revealed unsuspected cancer, atrophy of the stomach, or parasites, such as the anchylostoma. And yet many cases of anæmia of the most severe and often fatal type are observed where no organ or tissue is found diseased except the blood-making organs. To this disease the name pernicious anæmia is applied.

The malady is usually slow and gradual in its onset. The languor, pallor, tinnitus, dizziness, gastric and intestinal disturbances are the same as those noted under the head of chlorosis, but are usually exaggerated. Syncope is of frequent occurrence; breathing is rapid and there is a hunger for air; the heart's impulse may be felt over an increased area; the pulse is rapid, but strong and quick; the spleen is at times large; the bones may be sensitive on pressure. Hemorrhages are not infrequent, and retinal hemorrhage is rightly looked upon as of great diagnostic importance in this affection. There is progressive failure of strength. The patient becomes so weak a· to be unable to leave the bed; rising to the erect posture will frequently cause fainting. The œdema may become universal. The preservation of the adipose tissue is a striking feature of most cases of pernicious anæmia. Fever is usually present, the temperature seldom exceeding 103°. Death usually occurs inside of one year, yet improvement and even recovery are noted. Relapses are common.

The **blood** to the naked eye is pale and watery. Oligocythæmia is present in an extreme degree. The number of corpuscles may be less than 500,000. Poikilocytosis is marked. Megalocytes and microcytes are abundant. A striking characteristic of pernicious anæmia is the presence in the blood of nucleated red blood-corpuscles. These, when of the size of normal red blood-globules, are spoken of as **normoblasts**; when very large, as **gigantoblasts.** Ehrlich regards the **gigantoblast,** when present in the blood, as a proof of the pernicious character of anæmia. The white corpuscles are not absolutely, though often relatively, increased in number. There may be an increase in the small lymphocytes at the expense of the polynuclear elements. The total amount of hæmoglobin is less than normal, but the corpuscles are individually rich in hæmoglobin, so that, considering the number of red blood-corpuscles, the percentage of hæmoglobin is large.

FIG. 43.



Blood of progressive pernicious anæmia. Shows large and small red blood-corpuscles (macrocytes and microcytes,) with marked poikilocytosis. The large red blood-corpuscle just below the centre of the field, with excentrically placed nucleus, is a gigantoblast. (RIEDER.)

From chlorosis the disease is differentiated by the greater severity and progressive character of the symptoms, the greater tendency to hemorrhage, by the relative increase in the hæmoglobin, and the presence of gigantoblasts. From secondary anæmia it is to be distinguished by the blood findings and by the absence of the evidence of disease in any organ.

SECONDARY ANÆMIA.

Secondary anæmia may be the result of a hemorrhage, either acute or chronic; of starvation, as in cases of infantile marasmus, and of œsophageal obstruction; or it may be the consequence of almost any severe disease, whether acute or chronic. Chronic intoxication by lead, mercury, and chronic malaria will produce severe anæmia. It is usually attended by emaciation and weakness. It is thus found in connection with cancer, with suppurative disease, with tuberculosis, Bright's disease, and following the acute infective fevers. In certain secondary anæmias there is evident impairment also of the blood-making organs, so that the anæmia may assume almost a primary importance. Thus in a case of carcinoma with marked anæmia, Strümpell found secondary carcinosis of the bone marrow. An examination of the blood in doubtful cases should always be made. The blood in secondary anæmias usually shows oligocythæmia, oligochromæmia, the percentage of hæmoglobin being relatively less than that of the red corpuscles, moderate poikilocytosis and leucocytosis, rarely nucleated red corpuscles.

It may be of service to represent in tabular form the findings in typical cases of chlorosis, secondary anæmia, and pernicious anæmia. It should be understood, however, that there are many variations from the type, and, as already said, in many cases it is difficult to draw the line clearly between the various forms of anæmia. These figures are, of course, merely illustrative:

CHARACTERISTICS OF BLOOD IN THE ANÆMIAS.

	Normal blood.	Chlorosis.	Secondary anæmia.	Pernicious anæmia.
Red corpuscles	5,000,000	4,000,000	3,000,000	800,000
Hæmoglobin	90–100	30	60	30
Leucocytes	5,000	5,000	10,000	5,000
Microcytes and macrocytes	0	Few	Few	Many
Normoblasts	0	0	Few	Usually many
Gigantoblasts	0	0	0	Usually many
Poikilocytes	0	Few or many	Few or many	Many

SCURVY.

Scurvy is a disease that is caused by the lack in the blood of those materials supplied particularly by fresh vegetable foods. It is thus seen especially on shipboard or in almshouses or barracks, where vegetable food cannot for a long time be obtained. The absence from the diet of other articles than vegetables has been said to produce scurvy. It has been observed in Russia after long fasts where there has been a lack of animal food.

The disease comes on slowly with the symptoms of debility and anæmia. There are loss of strength, some derangement of the appetite, cardiac irritability, and usually quite early, the characteristic changes in the gums. The **gums** become swollen, of a purplish-red color, spongy, and bleed upon the slightest provocation. There may be loss of tissue and genuine ulceration. The teeth may become loosened in their sockets, and in some of the severer cases there is actual necrosis of the jawbone. The odor from the breath is very offensive. There is great pain on movement of the mouth, as in talking, mastication, and swallowing. Largely through the difficulty in mastication and swallowing the patient takes an insufficient amount of food, so that for this reason there is loss of strength and emaciation.

The **hemorrhagic tendency** of the disease is shown not alone in the bleeding from the gums, but in hemorrhages from other mucous membranes. Thus epistaxis and—though rarely—hæmoptysis, hæmatemesis, hæmaturia, may be present. Subcutaneous **ecchymoses** may develop spontaneously or as the result of slight traumata. Hemorrhagic infiltration of the muscles is quite characteristic. In the subcutaneous tissue there may be, following a hemorrhage, the development of connective tissue, which, by its contraction, may produce deformity, especially when the hemorrhage is in one of the extremities near a joint.

The skin of scorbutic patients is usually dry, and seems predisposed to the development of furuncles. Œdema may be present as the result of pronounced anæmia. Constipation is the rule. The urine is variable in quality, usually of a high specific gravity. Headache and neuralgic pains in other parts of the body are frequently complained of. Mentally the patient may be bright, though he is at times quite dull, even delirious, and in severe cases may develop convulsions. Fever is not constant; in fact, it is frequently absent, though slight causes, *e. g.*, the neuralgic pains just referred to, seem to produce an elevation in temperature. Complications may also produce pyrexia. The peculiar phenomena of day and night blindness are occasionally noted.

Improperly fed children suffer at times from a cachexia regarded as scurvy. (Barlow's disease.) In these cases, which appear oftenest in rachitic children, there are noted, as in scurvy, a swelling of the gums, which may be spongy or the seat of ecchymoses, and the improvement of the patient on an anti-scorbutic dietary. If the teeth have not yet developed the gums may be unaffected. The lower limbs are peculiarly affected, being paretic, swollen, or with shiny, tense skin. They are oftentimes quite tender, and there is greater liability to fracture of the bone or to epiphyseal separation than in the case of adults, though even in adults, necrosis of bones or the disunion of old fractures and the reopening of old sores, are occasionally noted. Intra-muscular and subperiosteal hemorrhages are common.

The four facts upon which a diagnosis of scurvy is to be based are:

1. Improper diet.
2. Involvement of the gums.
3. Infiltration of muscles.
4. Rarity of hemorrhages from lungs, stomach, bowel, bladder, kidney.
5. Lack of high temperature and septic appearance, such as is common in purpura hemorrhagica.

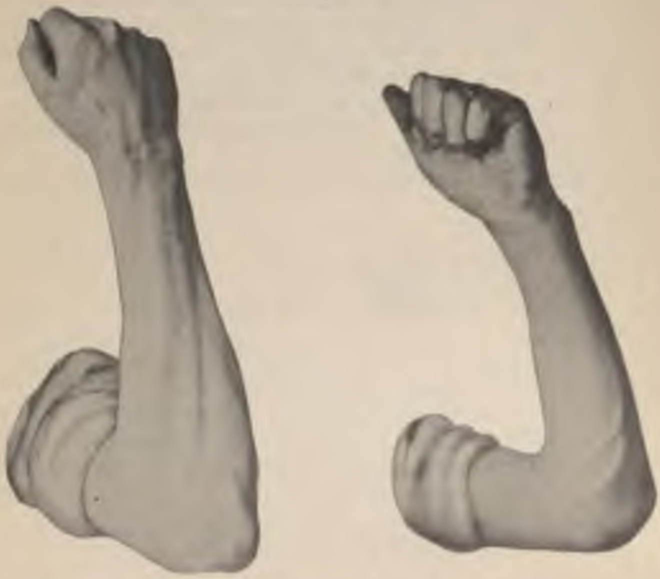
The prognosis in cases of scurvy is, now that the disease is well understood, excellent. Death may occur, however, where the disease is not early recognized, or where some complication, such as pneumonia, intervenes.

HÆMOPHILIA.

Hæmophilia, or the bleeder's disease, is a hereditary affection in which there is a tendency to uncontrollable bleeding from slight wounds or from no assignable cause. It is a peculiar feature of this disease that the transmission of this tendency is almost exclusively through the female, who, though the daughter of a bleeder, may never during life give any evidences of the hemorrhagic tendency. Males are bleeders much more frequently than females. No definite anatomical peculiarity has been described, and it is uncertain to-day whether the disease depends upon some abnormality in the structure of the vascular system, or upon alteration in the composition of the blood by which coagulation in a wound is prevented. No uniform changes in the blood have been noted.

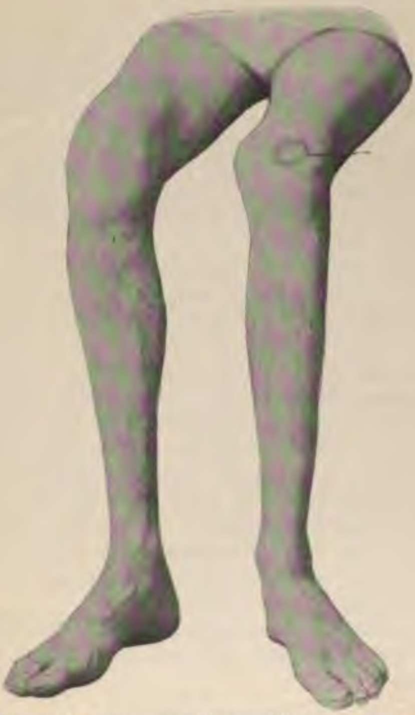
Hæmophilia usually manifests itself within the first two years of life. A slight cut or scratch may be followed by an uncontrollable bleeding that may result fatally or only cease upon the induction of syncope. Many patients, notwithstanding frequent and severe hemorrhages, live to adult life. The longer the patient survives, the better is the outlook as to life.

FIG. 44.



The hemorrhages not only occur when there is a traumatism involving the skin, but when there is any break in the mucous membrane. Frequently the hemorrhage is spontaneous, and from no assignable cause. Hæmoptysis or epistaxis will thus occur. Blood may be vomited or passed at stool, or may appear in the urine. A slight blow, or pinching of the skin will be quickly followed by an effusion of blood beneath the skin. Hemorrhages not infrequently occur into the joints. These joint hemorrhages are often associated with most severe pains, often described as neuralgic or rheumatic. At these times the joints are frequently swollen, tense, and oftentimes discolored by the blood that is effused into them. These arthritic pains may occur also without the evidence of hemorrhagic effusion into the joint. Permanent deformities of the joint may result, probably from the development of connective tissue following the hemorrhages. (Figs. 44 and 45.)

FIG. 45.



Figs 44 and 45 show joint changes of hæmophilia. An attempt was made by the patient to flex the arms, clench the fists, and make complete supination. This is seen to be but partially successful, owing to anchylosis. The deformity of the left knee is plainly seen; the foot was everted, flexion was limited. The shaded circle over the left knee represents the situation of the patella.

The diagnosis of the disease is rarely difficult where we have a clear family history. The disease must be, of course, differentiated from the various varieties of purpura, from scurvy, and from the hemorrhagic tendency that sometimes accompanies the acute infectious diseases. Of great value in establishing the diagnosis are the history of the family tendency, the repeated hemorrhages, and the evidence of permanent joint changes.

PURPURA.

Purpura hemorrhagica is a term used to describe a hemorrhagic diathesis that is not hereditary, as in hæmophilia; not due to any dietetic error, as in scurvy, but is acquired. In this disease there are extravasations of blood into the skin, petechial when they are small, ecchymotic when larger. No blood change has yet been discovered to account for these lesions. The blood probably escapes by diapedesis or by a rupture of the vessel wall.

Purpuric lesions may be **symptomatic.** Thus petechiæ are observed in severe cases of the exanthematous fevers, as smallpox, measles, scarlet fever; in pyæmia and septicæmia they are also noted. Purpuric lesions may also be due to cutaneous **emboli,** as, for instance, in cases of malignant endocarditis. Certain **drugs,** as the iodides and belladonna, may produce purpura; and in **cachectic states,** as in cancer, tuberculosis, Bright's disease, senility, the ecchymotic spots are frequently observed, especially upon the extremities. In certain neuralgic affections, in some cases of cord disease, and rarely in the hysterical, cutaneous extravasations have been seen; and it is not to be forgotten that rupture of a blood-vessel may occur from mechanical causes, as in asthma, emphysema, epilepsy, and pertussis.

As a distinct affection purpura is usually divided into three varieties—**purpura simplex, purpura rheumatica,** and **purpura hemorrhagica.**

Purpura Simplex

is accompanied by very slight constitutional disturbance; at times a slight fever, a little malaise, some diarrhœa, possibly swelling and pain in the joints. No hemorrhages are noted save the cutaneous, and chiefly into the skin covering the lower extremities. Recovery ensues in from one to two weeks.

Purpura Rheumatica.

Purpura rheumatica (peliosis rheumatica or Schönlein's disease) is in many cases apparently a rheumatism, with urticarial wheals, and, later, purpuric lesions. The lesions resemble closely those of erythema nodosum, with which they are often confused, and from which they cannot always be differentiated. Whether the arthritis is a genuine rheumatic arthritis is not settled. Preceding or accompanying the appearance of the lesions, which are usually found upon the lower extremities, there are sore-throat and malaise. The temperature ranges from 100° to 103°. The joints are usually painful, distinctly swollen; endo- and pericarditis have been noted. The prognosis is good, yet relapses, in some cases occurring yearly, are common.

Purpura Hemorrhagica.

Purpura hemorrhagica, or **morbus maculosus Werlhofii**, resembles in many respects an acute infectious disease, and it is highly probable that a bacillus (Letzerich) is the cause of the affection. It is a distinct and independent disease, unlike scurvy and hæmophilia. It is self-limited. While often so severe as to prove fatal, cases of recovery have been observed. It is often met with in delicate individuals, and yet may attack adults who are enjoying perfect health.

Some debility and malaise may precede for a few days the appearance of the purpuric spots. These spots make their appearance in the skin of various portions of the body, and range in size from the most minute up to those several inches in diameter. Bleeding occurs also from, or into, the mucous membranes. Thus there may be conjunctival ecchymoses or hemorrhagic areas in the mucous membrane of the mouth. Hemorrhage from the nose, the mouth, the stomach, the kidney, bladder, or intestines is not unusual. Death may result from hemorrhage into the brain. It is rare for hemorrhage to occur into the serous cavities in this disease, though in scurvy such hemorrhages are common. The fever of morbus maculosus is quite variable, in some cases being distinctly intermittent, in others remittent, or even pursuing a very irregular and erratic course. The patient may sink quite rapidly into a typhoid state, the pulse becoming weak, the mind clouded, the gastro-intestinal tract deranged, the fever ranging from 100° to 104°. Delirium may be prominent. Hysterical manifestations are sometimes observed. In severe cases the spleen is often enlarged. Death occurs in these cases from progressive debility, from acute anæmia the result of repeated hemorrhages, or apparently from septicæmia.

This disease must be differentiated from symptomatic purpura, which has been spoken of before, and which is usually clearly recognized by the symptoms characteristic of the primary affection. Purpuric lesions due to emboli should also be excluded. From hæmophilia it can be distinguished by the fact that the latter disease is hereditary, is usually found in males, is distinctly chronic in its course, has the peculiar joint involvements, and lacks the clinical history of an acute infectious disease, with fever, malaise, rapid pulse. It is unlike scurvy in that there is no history of deprivation of vegetable food. There are, also, no peculiar changes in the gums, and amelioration of symptoms does not follow the administration of antiscorbutic remedies. Hemorrhages from the lungs, stomach, kidney, all are common in purpura hemorrhagica. Morbus maculosus is neither endemic nor epidemic.

HÆMOGLOBINÆMIA.

In some manner not clearly understood, the hæmoglobin may escape from the red blood-corpuscles into the plasma of the blood, constituting the condition known as hæmoglobinæmia. The small amount of hæmoglobin that has thus escaped may be taken care of by the liver and spleen and give no symptom by which the condition can be recognized. Where larger amounts have escaped, the coloring matter of the blood passes off in the urine, constituting hæmoglobinuria.

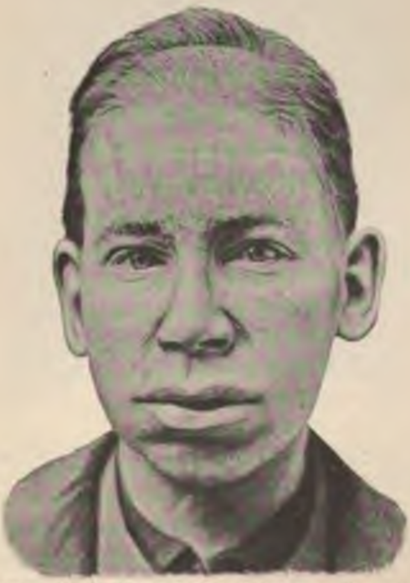
Paroxysmal hæmoglobinuria is characterized by attacks in which, with fever and nervous phenomena, the urine is voided containing the blood-coloring matter, but not blood corpuscles—at least, blood corpuscles not in any large amount. The exact etiology of this rare affection is unknown. At times malaria, syphilis, psychic influences, muscular strain, or a chilling of some portion of the body seem to be the exciting causes. It is associated at times with Raynaud's disease; at times with menstruation. Children and adults are equally attacked, oftener the male sex. There is usually an initial chill, a rise in temperature of two to four degrees; the pulse is much increased in rapidity, often reaching 130 or 140; the patient complains of pain in the back or in the extremities. The countenance may become pale or cyanotic, and icterus is frequently noted. Tenderness on pressure may be discovered over the region of the liver, kidney, or the spleen. The urine contains albumin and the coloring-matter of the blood. Blood corpuscles are absent or present in very small numbers, usually appearing broken or decolorized. Such an attack may last but a few hours or a few days.

Recurrences may be numerous; daily occurrences have been noted. The symptoms may be so slight as not to alarm a patient unless he accidentally discovers the bloody color of the urine. In other instances the symptoms are severe, and the patient is so prostrated as to take to his bed. Examination of the blood shows the plasma of a more reddish tint than normal, and pale, small, red globlues swimming in this fluid.

HÆMOGLOBINURIA OF THE NEWBORN.

Hæmoglobinuria of the newborn, as observed and described by Winckel, appears epidemically, from unknown causes, in children about the fourth day after birth. The child becomes cyanotic, icteric, the respiration is increased, pulse rapid, skin cool, temperature slightly elevated; vomiting and diarrhœa are present. The urine contains hæmoglobin, some albumin, granular casts, and a few blood-corpuscles. Death usually occurs with convulsions.

FIG. 46.

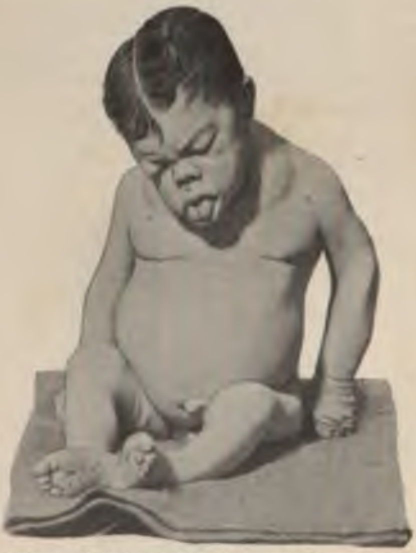


Myxœdema. (C. W. Burr's article in Dercum's *Nervous Diseases.*)

MYXŒDEMA.

Myxœdema is a term used to describe the development of a sort of myxomatous growth, rich in mucin, in the subcutaneous connective tissue. The frequent association of myxœdema with atrophy or destruction of the thyroid gland, and the improvement in the condition under the administration of thyroid extract, lead to the conclusion that there is some causal relation between thyroid destruction and myxœdema. It may possibly be that the active thyroid renders inert certain substances that now, when the thyroid fails to perform its function, accumulate in the body and produce the peculiar change characteristic of myxœdema.

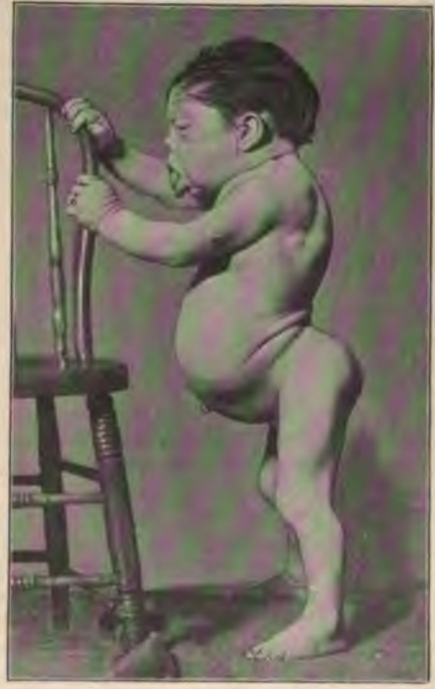
FIG. 47.



Cretinism. (Burr's article in Dercum's *Nervous Diseases.*)

The myxomatous condition is most plainly noted in the face. The skin appears thick and swollen, the wrinkles disappear, giving to the face a dull, stoical look. In other parts of the body the same change may be noted. The tongue may be thickened, the ends of the lower extremities also, though usually in less marked degree than is the face. While the skin feels hard and resistant, it is to be noted that there is no pitting on pressure, as in the case of true œdema. Various trophic disturbances, as the loss of nails and hair, dryness of the skin, are frequently found, and sensory anomalies are also noted. As the disease progresses mental weakness becomes more and more pronounced. The patient becomes apathetic, and the case may present ultimately the picture of terminal dementia. The temperature in myxœdema is usually normal or subnormal. Albumin and sugar are occasionally found in the urine.

FIG. 48.



Cretinism. (BURR's article in Dercum's *Nervous Diseases.*)

The prognosis is always grave, though patients may live for from five to fifteen years. Women are oftener affected than men.

Sporadic cretinism, or congenital myxœdema, is a term applied to a form of mental weakness, idiocy, or imbecility that is congenital, or develops in early infancy, and is associated with alterations in the thyroid gland. These patients are poorly developed dwarfs, with large faces, thick lips, and prominent abdomens. Umbilical hernia has been noted in many cases. They are slow to walk, slow to talk, or may never learn to talk intelligibly.

The **operative** form of myxœdema is that form which develops following the extirpation of the thyroid gland.

Myxœdema should not be confused with the œdema of Bright's disease. The pitting on pressure, the characteristic urine, the cardiac changes, and the evidences of uræmia enable the differential diagnosis to be made. Considerable difficulty in deciding between scleroderma and myxœdema may be encountered. Here, however, the absence of mental dulness, and the hide-bound condition of the skin make the diagnosis clear.

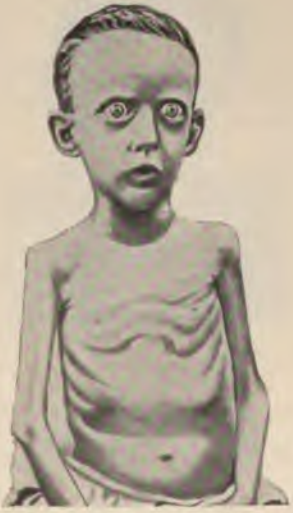
EXOPHTHALMIC GOITRE.

In typical cases of exophthalmic goitre the disease is readily recognized by the presence of the classical trio of symptoms—exophthalmos, goitre, and cardiac irritability. In not a few cases, however, one or more of these classical symptoms may be absent. It is characteristic of exophthalmic goitre that the entire nervous system seems to be in a state of irritability. Slight disturbances are followed by disproportionately marked effects.

Exophthalmos, or prominence of the eyes, may appear early or late. It is usually symmetrical, rarely single, and is due to fulness of the orbital bloodvessels and to the exposure of the eye by the retraction of the lids. There is often found an inco-ordination of the lid and globe in looking downward, the lid movement being retarded and the lid held retracted (von Graefe). When marked, exophthalmos gives to the patient a wild, staring, frightened expression that at once draws our attention to the nature of the disease. The eye movements may be quite limited in extent in all directions. This is especially seen in the voluntary effort at convergence for near vision, as where the patient follows the movements of the finger that is brought closer and closer to the patient's nose. Nystagmus, corneal anæsthesia, and ulceration may occur. Dilated and inactive pupils are also noted at times.

Rapidity of the heart's action, increase in its strength, and irregularity in its beat, are prominent characteristics of exophthalmic goitre. The tumultuous action of the heart is often plainly perceptible to the patient, and to inspection or palpation on the part of the physician. The larger arteries are also seen to pulsate violently. Very slight emotional or nervous influences disturb greatly the cardiac regularity. Heart murmurs may be due to valvular lesions, which not infrequently are present as complications. Cardiac hypertrophy is not uncommon.

FIG. 49.



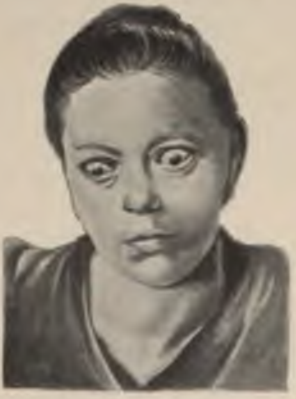
Exophthalmic goitre in a child, aged three and a half years. All the symptoms present except tremor. Case of Drs. Renshaw and Dreschfeld. (From BURY.)

The **thyroid gland** is usually enlarged in the lateral lobes, in the isthmus, or in both. It may be somewhat hard, firm, and unyielding from the abundance of fibrous tissue, but is oftener elastic and pulsating, with a perceptible thrill or a systolic murmur. Goitre may, however, be absent.

The next most common symptom is **tremor**. These tremors may be fine, and represent five to ten oscillations per second. They may be very pronounced or barely perceptible, and much increased by excitement or voluntary effort. They are most plainly observed in the upper portion of the body. There may be such marked trembling as to resemble paralysis agitans or even chorea.

Muscular cramps have been noted as well as paraplegia. Hysteria may complete the disease, and may be the cause of the paralysis, cramps, anomalies of sensation, and disturbances of vision occasionally observed. The urine is frequently increased in amount, and this, even in patients not demonstrably hysterical.

FIG. 50.



Exophthalmic goitre. Von Graefe's sign. (OPPENHEIM, after BRUNS.)

Among the diseases that have been noted as associated with Graves's disease are tabes, diabetes, and myxœdema. The menses are usually scanty; the genitalia may be atrophied, the mammary gland reduced in size. The digestive function is oftentimes impaired, the appetite being deranged, and nausea, vomiting, diarrhœa, not infrequent. The blood shows a moderate degree of anæmia. There is usually loss of flesh and of strength. The vasomotor nerves are seen to be impaired by the red flushing of the skin. The skin may also be pigmented and resemble that of Addison's disease. Excessive perspiration is common.

The diagnosis cannot be made upon the presence of any one symptom. It is the peculiar combination of symptoms that enables the diagnosis to be made. The disease may occur with great acuteness in the course of a single day, or it may come on very insidiously, so that the patient cannot tell when the disease really began.

Among the causes assigned are fright, grief, severe illness, parturition. Heredity, too, seems to play some part.

ADDISON'S DISEASE.

Addison's disease, probably due to lesions of the sympathetic nervous system, and, in the majority of cases also, of the supra-renal capsules, is characterized by bronzing or pigmentation of the skin, great asthenia, feeble heart action, vomiting, and diarrhœa. The disease may occur at any age, though it is commonest in adults. Sixty per cent. of all cases are found in males. The disease is usually chronic, lasting from six months to as many years, though it may prove fatal in from two to three months.

The most characteristic symptom is the **pigmentation** of the skin. This is first noticed, and is most marked, on the face and hands and in those situations where pigment is normally somewhat more abundant, as about the nipples, in the axillæ, and on the scrotum. The hue varies from a slight brownish discoloration to a deep brown or even black color. This pigmentation may cover the entire body, though patches may be found where the hue is much darker than in other places, giving the body a somewhat mottled appearance, much as in vitiligo. Slight variations in the tint of the skin may be noted at different times in the same case. The symptoms and post-mortem lesions of Addison's disease have been observed in cases where pigmentation was very slight, or, it is said, even lacking. These cases have run an acute course.

The **pulse** of Addison's disease is small, feeble, compressible, and, late in the history of the malady, quite rapid. Heart lesions are seldom found to account for the altered pulse beat. Cerebral anæmia, with attacks of dizziness and syncope, is frequently noted. The **asthenia** is usually pronounced, and may be one of the earliest symptoms. Death in these cases often occurs without premonitory warning, apparently from syncope or sudden cardiac failure. There is usually a moderate degree of **anæmia**, the blood showing some diminution in the red corpuscles and in the amount of hæmoglobin; rarely, however, do the red globules diminish below 2,000,000. In most cases of Addison's disease there are **gastro-intestinal disturbances**; constipation, which is sometimes noted at the beginning, is usually replaced by diarrhœa later, and this is oftentimes associated with intractable vomiting. It is characteristic that the alimentary disturbances do not follow errors in diet. **Pain** has been noted in one-third of the cases of this affection. It is usually described as sharp and neuralgic, or oftener of a severe, dull, aching character, and is referred chiefly to the lumbar region or to the epigastric and hypochondriac regions. Late in the course of the disease **mental weakness** and lack of energy are noted. Delirium or coma may precede a fatal result.

In typical cases with distinct bronzing of the skin, unaccountable vomiting and diarrhœa, feeble, rapid pulse, great weakness, mental hebetude, localized lumbar and epigastric pain, the diagnosis is readily made. Where the pigmentation is very scanty other causes for the asthenia, feeble pulse, etc., must be excluded. And it is to be remembered that pigmentation of the skin may be quite marked in those exposed to the sun's rays, in brunettes, in certain races of people, in pregnant women, in tramps and vagabonds through the irritation of lice and dirt, in tinea versicolor, and in some cases of hepatic disease and abdominal growths, especially tuberculosis of the peritoneum. In cases of melanotic cancer and also of exophthalmic goitre, abnormal pigmentation of the skin has been noted.

RICKETS.

Rickets is a disease of children, characterized by malnutrition and deformities of the bones. It is most frequently observed at the period of first dentition, though the disease may not show itself until as late as the period of the second dentition. The exact causes of rickets are not yet fully determined. Among the etiological factors must be mentioned improper diet. Well-fed and well-nourished children are rarely the subjects of rickets. Heredity seems to play a not unimportant part in the development of rickets. The children of tubercular and syphilitic parents seem especially prone to the nutritional errors characteristic of this disease. It is oftener observed in cold and damp countries. Artificially fed children are much oftener affected than sucklings.

The disease may come on insidiously, and nothing wrong may be noticed by the parents until the time when the child should begin to walk. It is then seen that the child makes little or but feeble effort to walk, and that there is an inclination to deformity of the lower extremities. In other cases, preceding the deformities of the bones, there are slight fever, irritability of temper, restlessness, with gastro-intestinal disturbances.

Profuse sweating is a very common phenomenon in rickety children. The child does not present the plump, rosy appearance of health, but soon becomes puny, feeble, and very much weakened.

The deformities of the bony structures, constituting the most characteristic features of the disease, are various. The head of a rickety child usually shows a broad, square forehead with prominent frontal eminences. The fontanelles are slow to close, and areas of incomplete ossification are frequently found which yield readily to the pressure of the finger. To this condition the name **cranio-tabes** has been applied. The resemblance between the enlarged head of rickets and that of chronic hydrocephalus is quite striking, and yet in the case of hydrocephalus the mind is usually dull, while in the case of rickets the child may be bright and intelligent. The bones of the face are often deformed, the angle of the lower jaw approaching a right angle, the anterior portion of the upper jaw being prominent. The enamel of the teeth, which often appears late, is frequently eroded, and the second teeth may crumble and decay. Various deformities of the spinal column—anterior, posterior, and lateral—are seen in rachitis. The sternum is often short, strongly arched forward at its middle, producing the form of chest known as pigeon-breast. Bead-like prominences at the junction of the ribs and their cartilages, due to osteo-chondritis, may produce the rachitic rosary on the chest. The deformities of the pelvis may not be noted in early life, but in women at the time of childbirth they may be the cause of great interference with parturition. The bones of the lower extremities are peculiarly liable to alteration, because they are obliged to carry the weight of the child and yield readily to pressure. Numerous cases of knock-knee and of bow-legs owe their origin to this nutritional disease of bone. Fractures occur quite readily in rickety bones, being often of the green-stick variety.

The diagnosis of this disease is not at all difficult in typical cases. Mild forms of the disease are to be suspected when there is an irregular development of the teeth, when there is chronic diarrhœa without dietetic error to account for it, nervous irritability, copious perspiration. The deformity of the skull of rickets is to be differentiated from that of chronic hydrocephalus by the fact that the latter disease is usually accompanied by symptoms of irritation, as convulsions, and by marked mental weakness. The deformities of osteomalacia are not observed among children except very rarely. The deformities of congenital syphilis are to be recognized by the characteristic lesions of that disease.

OSTEOMALACIA.

The peculiar and not clearly understood softening of bones, known as osteomalacia rarely attacks children. Adults in certain localities, *e. g.*, northern Italy, Westphalia, Flanders, particularly women who have borne children, and those with poor hygienic surroundings, are most prone to be attacked.

Pain in the bones, as in the sacrum, spinal column, pelvic bones, is an early and persistent symptom. Later there may be exquisite bone tenderness. Soon the softening of bones permits of their distortion. We thus find the spinal column showing various curvatures, the chest changing in shape, the lower extremities yielding. In this way there is often great deformity and a lessening of the stature. Pelvic examination shows deformed pelvis. The ribs and long bones can be readily bent. The functions of most of the organs are well performed, but from the pain and great difficulty in walking the patient finally becomes bed-ridden. Complications, *e. g.*, pneumonia, general debility, usually cause death at the end of a few years.

Before bone softening is marked the disease may be mistaken for spinal dicase. The peculiar hobbling gait, the absence of other evidence of spinal disease, as altered reflexes, and, if necessary, the watching of the case until the softening and distortion of the bones are manifest, enable the physician to make a diagnosis.

Rickets is a disease of childhood, does not have the bone pains. The changes in the skull are marked in rickets, absent in osteomalacia. Epiphyseal thickenings, exemplified by the "rosary," are common in rickets, not found in osteomalacia.

DIABETES MELLITUS.

Diabetes mellitus is a disease characterized by the passage of a large amount of saccharine urine. It is to be distinguished from the transitory appearance of sugar in the urine constituting glycosuria. Though diabetes has been recognized for years, but little is known of the exact etiology of the disease. It is due in certain cases to disease or injury of the brain, particularly the floor of the fourth ventricle. Severe mental strain and overwork may produce it. Disease of the liver is assigned as a cause, and lately much attention has been given to the connection between disease of the pancreas and diabetes, as in many cases of saccharine diabetes pancreatic disease has been recognized during life or post-mortem. It is most common in adult life, although it has been observed in infants. It is oftener met with in men than in women, and Jews are in some way peculiarly liable to the disease. Obesity and diabetes are frequently associated.

The characteristic feature of diabetes mellitus is the passage of a large amount of urine that contains sugar. This is proof positive of the disease, when it can be shown that the sugar is constantly present in the urine. The urine of diabetes differs from normal urine in its amount, specific gravity, and in its containing sugar. The amount is commonly increased, varying usually from four to twenty pints. Rarely, the amount will be practically normal. The specific gravity is high, seldom being less than 1025, and at times reaching 1050. The color of the urine is pale, the odor sweetish, the taste saccharine. It is usually acid in reaction. The amount of sugar may vary from a trace to from 5 to 10 per cent.

There are many tests for sugar that are quite reliable. The more important of these tests are described under the head of "Examination of the Urine," page 281.

In diabetic urine the urea and phosphates are usually increased. Albumin is occasionally found.

The first thing to attract the attention of the patient is usually the fact that he is passing a large quantity of urine; that he rises at night five or six times in order to empty the bladder. Associated with this is an unquenchable thirst. It may be that the whitish spot or stain, left upon the night clothing or upon the body linen when the urine has evaporated, attracts his attention; or he may be annoyed by the itching or irritation of the skin about the genitals. In other cases the disease comes on so insidiously that the patient is unaware that he is ill, until he applies for life insurance, and has the urine tested by the life-insurance examiner, who finds sugar.

As the disease progresses there are great loss of strength, and emaciation. The skin is usually harsh, dry, oftentimes the seat of furuncles or of carbuncles. It is characteristic, too, of diabetes that slight wounds or bruises are slow to heal. The appetite of diabetics is oftentimes good, even excessive. Patients with diabetes not infrequently suffer from dilatation of the stomach, because of the excessive amount of food that is daily ingested. The tongue is usually dry, red, and the saliva scanty. Constipation is the rule; fever in uncomplicated cases is rare.

Among the complications may be mentioned the boils and carbuncles spoken of above, itching of the skin, and the occurrence of gangrene. Fifty per cent. of patients with diabetes are affected with pulmonary tuberculosis. Acute croupous pneumonia, not infrequently followed by gangrene, destroys a large percentage. Among the nervous complications should be mentioned the neuralgias, particularly sciatic neuralgia, which is so frequently bilateral. Diabetic patients are oftentimes morose, melancholic, or hypochondriacal. The sexual function is depressed, and impotence is frequently an early symptom. The patellar-tendon reflex is often absent. The sight may be impaired, either from a retinitis, hemorrhages, cataract, or from causes the anatomical basis of which is not clearly understood.

Many patients with diabetes die comatose. Diabetic coma resembles in many particulars uræmic coma. This coma may develop suddenly, with syncope, drowsiness, and complete stupor. Oftentimes there is a preliminary gastro-intestinal disturbance. Dyspnœa, with rapid respiration, delirium, and headache may precede the fatal coma. It is in these cases that there is observed the heavy, sweetish odor of the breath due to the presence of acetone. This odor of the breath may occasionally be observed in diabetics without the occurrence of coma. Acetone and diacetic acid may be found in the urine, but do not necessarily presage an attack of coma.

As regards the course and prognosis of diabetes, it is to be remembered that as a general rule, the younger the patient the more unfavorable the prognosis. Many cases occurring in elderly, fleshy individuals pursue a benign course, the patient living many years, dying perhaps of some other disease.

The diagnosis of diabetes is established beyond a question by the examination of the urine. Transient glycosuria is to be eliminated by repeated examinations. Among the symptoms that should lead the physician to examine the urine for sugar, should be mentioned polyuria, excessive thirst, dryness of the skin with a tendency to carbuncles or furuncles, eczematous eruptions and pruritus about the genitals, cataract, emaciation, and weakness without any assignable cause. A bilateral sciatica should always lead to an investigation of the urine.

DIABETES INSIPIDUS.

Diabetes insipidus is a chronic disease in which large quantities of non-saccharine urine, of low specific gravity, are passed. Transitory diuresis or polyuria, as met with in hysteria, or as one of the phenomena of Bright's disease, is not diabetes insipidus. The malady is met with most frequently in the young, even in infants. In many cases the exciting cause seems to be some affection of the nervous system, sudden fright, blows upon the cranium, lesions of the brain, and of the medulla. In other instances abdominal tumors, abdominal aneurism, and other abdominal conditions that may influence the neighboring plexuses of nerves, seem to be the efficient cause. Cases in which there is a marked hereditary tendency are reported.

Ordinarily the disease manifests itself by a gradual increase in the amount of urine voided, and a corresponding increase in the amount of water drunk in response to the ever-present thirst. In some instances the onset is rapid. The general health may be very little interfered with; the patient may live for many years, and aside from the annoyance and inconvenience occasioned by the great thirst, and the frequent micturition, present no evidences of disease. In other instances there is more or less impairment of the general health, loss of flesh, and of strength, and death, usually from some intercurrent affection which readily attacks the individual, whose resisting power is thus materially lessened.

The diagnosis rests upon the examination of the urine and the absence of any renal disease to account for the increased amount. The urine is pale, of a low specific gravity, seldom exceeding 1007, contains no albumin, no sugar; the total solids may be present in normal amount, or even in increased amounts. Very rarely traces of sugar and of albumin are found. The amount of urine varies from fifteen to fifty pints in twenty-four hours. Some of the more anomalous symptoms of the disease are the subnormal temperature, disordered digestion, paralysis of the sixth nerve, loss of reflexes, retinal hemorrhage, and neuro-retinitis.

The disease is readily distinguished from diabetes mellitus by the low specific gravity and the absence of sugar; from hysterical diuresis by the transitory character of the latter affection and the occurrence of other symptoms of hysteria. Chronic Bright's disease may simulate it in the large amount of urine, with low specific gravity, that may be passed, but, in this condition careful examination will reveal albumin, though it may be but a trace, casts, and other evidences of renal origin of the polyuria, such as increased arterial tension, cardiac hypertrophy, œdema.

ACUTE RHEUMATISM.

Acute rheumatism attacks oftenest young adults and children, although it may be found in the aged. The larger number of cases are met with in the early months of the year. The disease is rare in tropical climes. Exposure to cold, sudden changes in temperature, heredity, have much influence in the production of this malady. While no specific germ has yet been proven to be the cause of the disease, there can be but little doubt that it is infectious.

Acute rheumatism may set in suddenly, or there may be premonitory malaise, headache, anorexia, or a slight chill. It attacks usually one joint at first, which becomes painful, tender upon pressure, red, and swollen. Fever is present, the temperature rising rapidly, even as high as to 104°. The pulse is usually soft and increased in rapidity. The tongue becomes coated, but usually remains moist. The urine is scanty, highly colored, strongly acid, of high specific gravity, and deposits upon cooling, urates; slight albuminuria may be noted.

It is characteristic of acute rheumatism that as the inflammation in one joint begins to subside, another joint, often the corresponding joint upon the opposite side of the body, becomes involved. In this way the disease may attack first the right knee, then the left one, then one elbow, then the opposite elbow; then an ankle, etc. Thus it may in turn attack many of the joints of the body, usually the larger ones. The swelling may involve not alone the joints proper, but some of the periarticular structures, or the sheaths of the tendons. The pain usually very severe. The slightest movement of the affected joint causes the greatest suffering. Profuse sweats are common, the perspiration being acid and of an acid, penetrating odor; sudamina are frequently noted upon the skin.

In acute rheumatism anæmia develops with great rapidity. Cardiac murmurs may thus be found that are not due to acute endocarditis, though it is to be remembered that among the commonest complications of acute rheumatism is acute endocarditis. The endocarditis is usually of a benign type, though it may be of the malignant variety. Other serous membranes, as the pericardium, the pleura, the peritoneum, may also be inflamed.

Among other complications should be mentioned **hyperpyrexia.** The exact cause of this is not known. The temperature may rise rapidly, the patient become delirious and present many other symptoms of meningitis. Preceding death the temperature may reach 100° or 110°. Delirium, coma, convulsions may be met with in some of the severe cases, and especially those associated with hyperpyrexia.

Chorea, especially in children, is not infrequently associated with rheumatism. Among the cutaneous complications may be mentioned purpura rheumatica and the scarlatiniform rash that are occasionally seen. The duration of the disease is variable. Many cases terminate in from one two weeks; in other cases, where joint after joint is successively involved, the disease may last for two or three months.

The acute onset of the affection, the inflammation of one joint after another, the scanty, acid urine, the acid sweats, render the disease very easy of recognition. Among the affections most likely to be confounded with it are the inflammations of joints occurring during the course of infectious diseases, as scarlet fever, cerebro-spinal meningitis, gonorrhœa; also the arthritis which is one of the manifestations of pyæmia. Osteomyelitis, occurring near a joint, should not be mistaken for acute rheumatism. Gout is recognized by its involvement of the joints of the great toe, and by the desquamation, less continuous fever, less copious perspiration, by the age of the patient, and the peculiar nocturnal attacks.

SUBACUTE RHEUMATISM.

Subacute rheumatism is a term applied to a form of the disease in which there is an involvement of a few joints, a mild course of fever, less intensity of pain—in short, a subacute course of the entire disease.

GONORRHŒAL RHEUMATISM.

The variety of arthritis that often occurs during the course of a gonorrhœa ordinarily goes by the name of gonorrhœal rheumatism, though it probably has no connection with true rheumatism, so that the name is a misnomer. At the height of the blennorrhœa, or when the urethral discharge has almost entirely ceased, a joint, usually the knee or the ankle, becomes swollen, reddened, tender, and exquisitely painful. Not alone is the joint itself inflamed, but the periarticular structures are œdematous and tender. The tendons and their sheaths, the adjacent bursæ, and the periosteum may also be affected. Rarely more than one joint is affected, and it should always excite the suspicion of an urethral cause, when a patient presents himself for treatment for rheumatism with but one joint involved. Among the rarer joints affected are the sterno-clavicular, temporo-maxillary, and sacro-iliac joints. Complications may occur with gonorrhœal rheumatism as with the ordinary rheumatism. Endocarditis, pericarditis, and pleurisy have been noted. The course of the disease is extremely slow—weeks, months, or years being necessary before the inflammatory action subsides. Anchylosis may occur as the result of this form of arthritis.

Its more sudden onset, the rapid involvement of periarticular structures, the œdema and purplish hue of the skin, the absence of tubercular history and of the evidences of tuberculosis in other parts of the body, with the history of an urethral discharge, serve to distinguish gonorrhœal arthritis from the tubercular form.

CHRONIC RHEUMATISM.

Chronic rheumatism occasionally follows as the result of an acute attack, but ordinarily originates as a chronic inflammation and with very few acute inflammatory symptoms. It is met with at a later period of life than is the acute form of the disease. Constant exposure to cold, residence in damp localities, poor hygienic surroundings favor the development of the disease. In this affection the joints are painful, perhaps slightly swollen and stiff. These symptoms are noted more particularly in the morning, after the rest of the night. During the day the joint seems to limber up and much of the pain disappears. Exacerbations are common. At these times there may be a slight temperature and the symptoms of acute rheumatism of a mild type. Such changes may occur in the joints and the tissues roundabout, and the joint may be more or less deformed. From constant suffering, from anæmia and malnutrition, the patients may really be in very poor health.

As regards life, the prognosis is good; as regards a cure, bad. Complications are rare. Valvular disease of the heart in chronic rheumatics is not ordinarily due to an acute endocarditis, but to the sclerotic changes incident to old age.

The disease is to be distinguished from gout and arthritis deformans. From the former it is distinguished by the absence of any history of the peculiar gouty paroxysms, and the absence of any uratic deposits in the neighborhood of the joint. The peculiar deformities of arthritis deformans with the exostoses, the contractures, and the tendency to primary involvement of the smaller articulations make the diagnosis of this disease usually easy.

MUSCULAR RHEUMATISM.

Muscular rheumatism, or myalgia, is a painful affection of the voluntary muscles which has received various names, according to its seat, **torticollis, lumbago, pleurodynia,** etc. It is found particularly in those who suffer from rheumatic affections of the joints, or who come from a rheumatic family.

The disease presents no constitutional disturbances; it is recognized merely by the symptom of localized pain. This pain may come on suddenly, as after a severe strain, exposure to cold, and especially sleeping in a draught when the body is covered with perspiration. The pain is oftentimes very severe, sharp and sticking in character, and the slightest movement of the affected muscle causes great suffering. Thus, a patient with lumbago, where the muscles in the small of the back are are affected, will lie motionless on the back, in order to be free from pain. So, where the muscles of the neck are affected, as in torticollis, the neck is carried stiffly with the head to one side.

In **pleurodynia** the intercostal muscles are involved, and efforts at respiration cause great suffering. This affection can readily be confused with pleurisy or with intercostal neuralgia. The absence of fever, of cough, and of the physical signs of pleurisy, should serve to exclude the former disease, and the absence of painful points on pressure, the latter. In fact, pressure over the affected muscles does not, usually, in muscular rheumatism excite pain.

Among other muscles that may be affected may be mentioned those of the œsophagus, stomach and intestines, the muscles of the abdominal wall, of the scalp, and those about the shoulders. The duration of the disease is most variable, some cases subsiding in a few hours, and others lasting for several days.

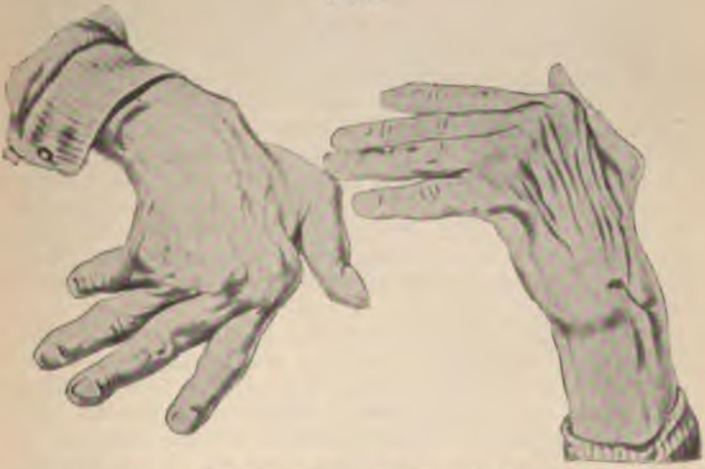
ARTHRITIS DEFORMANS.

This disease, oftentimes spoken of as rheumatoid arthritis, nodular rheumatism, or rheumatic gout, really seems to have no connection with either rheumatism or gout, but is probably of nervous origin. It is customary, however, to class it with the diseases of nutrition. "The symmetrical character of the lesions, the systematic atrophy of the muscles, the progressive lesions of the bones, the fulgurant pains, the rapid palpitation of the heart, and the trophic changes in the skin that are observed in nodular rheumatism, so closely resemble the corresponding lesions that accompany dorsal tabes, hemiplegia, paralysis agitans, exophthalmic goitre, and other nervous disorders, that many pathologists incline to the belief that their common cause must be sought in the nervous system. With regard to the nature of that cause, if it exists, it must be admitted that little or nothing is known." (Lyman.)

The disease is met with oftenest in young females, though it may occur in children. I have seen a typical case in a child of three years. In most cases no cause can be assigned. The joints most commonly affected at the beginning are the small ones. Attention is called to these by some pain, often quite slight, and swelling. There may be an onset of a subacute character in which several smaller joints are affected, the disease being ushered in with some fever and malaise. The swelling in these cases is not limited wholly to the joints, but it may be as well in the periarticular structures. Effusion into the joint seldom occurs. Gradually, other joints become involved, the larger joints, as the knees and hips, being involved last. The pain, when many of the joints are involved, may be extreme, usually worse at night, and yet in some cases pain is but a trifling feature of the case.

There is great difficulty in movement of the joints after the disease has become well established. This is due in part to the pain, but much more to the **contractures** of the muscles and the changes that take place in the cartilages and bones. Thus, in the fingers, the extensors or the flexors may be permanently contracted, producing peculiar alterations in the shape of the hand, which Charcot has well depicted. The fingers are often drawn toward the ulnar side. It will be seen, too, on examining the affected joints, that they seem much larger than normal, partly because of the **atrophy** of the neighboring muscles, but in a measure because of the overgrowth of bone near the joint. It is found on dissecting these joints that the cartilage has become destroyed over the centre of the joint, the subjacent bone has become eburnated, and the bone around is deformed by an *exostosis*. These alterations cause great limitation in the movement of the joint. Oftentimes, too, a crepitus can be felt, or a friction sound heard, on movement of the affected joints. The skin is shiny and glossy.

FIG. 51.



Arthritis deformans, showing distortion and muscular atrophy. (BURY.)

The number of joints involved may be very great, few being spared. In other cases, the disease seems to be checked when the joints of the hands and of the feet are involved; and there is a monarticular form found in old people, affecting the hip. To this form the name **morbus coxæ senilis** is applied. The disease always runs a chronic course, and is not incompatible with length of life. The amount of suffering and incapacity for work vary greatly in different cases. Exacerbations and periods of quiescence are common.

Typical cases can scarcely be confused with any other disease. The slow, insidious course of the disease, its predilection for the smaller joints, the osteophytes, the eburnation, as evidenced by crepitus, the peculiar deformities, make a picture presented by no other disease.

GOUT.

Gout is a disease most commonly attacking those in adult life who have been high livers, enjoying an abundance of rich food, especially meats, alcoholic drinks, particularly wines and beer. Males are oftenest affected, and hereditary influences are, in more than half the cases, clearly traceable. Lead poisoning is also assigned as a cause of gout.

Acute gout manifests itself by a peculiar paroxysm of pain, usually in the joints of the great toe of one foot. Premonitory restlessness, irritability of temper, gastric disturbance, and neuralgic pains, may be noted for some hours or days preceding the attack. The joint usually becomes inflamed during the night. The patient is awakened with sharp, excruciating pain, the joint is swollen, red, and extremely tender to pressure. There is fever, the temperature being about 102°. This paroxysm may last for a few hours only, to be repeated on the following night. The gouty attack may last for from three days to a week, the symptoms then gradually abate, and the patient may for a time feel much improved in his general health.

Following the inflammation of the joint there is local desquamation. Suppuration does not occur. In some cases, where there is a rapid abatement of the symptoms of local inflammation, more serious, general disturbances are noted. To this form of gout the term **retrocedent**, or suppressed gout, is applied. Among the severer symptoms may be mentioned diarrhœa, vomiting, great depression, cardiac irregularity, delirium, coma, collapse, or even death.

Where gout recurs frequently, it assumes a chronic form. The attacks may not be more severe, but last longer, and there are deposits of urates in the articular cartilages and in the tissues about the joint. With the subsidence of the acute attack the joint is now left more or less swollen and deformed. These deposits are usually found upon the feet, about the joints of the feet and hands, though they may be met with in the cartilages of the ear and of the nose. To them the name of **gouty tophi** has been applied. In some cases the skin over these chalk-stones may rupture and the gouty deposit be exposed to view.

In these cases of chronic gout there are also manifestations of constitutional disturbance that should be noted. There is frequently marked derangement of the stomach; gouty patients are notorious dyspeptics. They may be nervous and irritable. Arterio-sclerotic changes are prone to appear in these subjects. The pulse is of high tension, the left ventricle is hypertrophied, the kidney may become contracted; in short, we may have all the symptoms of a chronic interstitial nephritis. These patients are subject to headaches, neuralgic pains, muscular cramps, bronchial catarrh and asthma. The urine is usully high-colored, shows a deposit of urates, and is strongly acid. Where the kidney is diseased there will be albumin and casts, as in chronic nephritis from other causes. It was long ago noted that gouty patients frequently suffered from cutaneous eruptions, such as eczema.

DISEASES OF THE KIDNEY AND GENITO-URINARY ORGANS.

THE URINE.

The kidneys, from their anatomical location, are practically beyond the reach of inspection, palpation, and percussion, so that slight alterations in their size, shape, or degree of tenderness cannot be detected.

While physical examination of the kidney is not to be underestimated, we have to rely, in the diagnosis of renal disease, very largely upon the examination of the urine, of other organs, such as the heart, that can be secondarily affected, and upon the subjective symptoms of the patient. The examination of the urine is of such great importance that a brief *résumé* will be given of the methods of urinalysis. Only those methods that are of the most practical importance are here given. Other methods, and especially those for the quantitative analysis of the urine, can be found in some of the special works upon the urine. It is wise in examining the urine that some systematic plan should be adopted, such as the following:

URINARY EXAMINATION.

1. Quantity.
2. Appearance as to (*a*) color; (*b*) clearness; (*c*) deposits.
3. Odor.
4. Specific gravity.
5. Reaction.
6. Chemical examination. (*a*) Albumin; (*b*) sugar; (*c*) bile; (*d*) amount of urea; (*e*) peptone, indican, etc.
7. Microscopic examination.

amination the specimen of urine as it is voided in the morning,

Method of Obtaining Sample of Urine. For ordinary ex-
is all that is necessary. In other cases it is desirable that the specimen should be the urine passed during twenty-four hours.

To collect this, the patient should empty the bladder at a certain hour, say at seven o'clock in the morning, and reject the specimen then passed. All urine passed from this time to the same time on the following morning should be saved, including the urine passed at the close of the twenty-four hours, that is, seven A. M. This can be measured, and a sample of the mixture furnished the physician, or the entire quantity if desired, for special examination. It is desirable at times to have samples of urine taken before or after meals, before or after exercise. In other cases, when one wishes to obtain urine as free as possible from any urethral or vesical contamination, the catheter can be passed, the bladder emptied and washed out once or twice with a small quantity of warm water, and then, a catheter remaining in the bladder, the urine as it trickles drop by drop, is collected. This is practically the urine as it comes fresh from the kidney. Catheterization of the ureter, even in the male, has been resorted to as a means of obtaining urine from each kidney separately, where there is reason to suspect unilateral disease or the existence of but one kidney.

Physical Characters of Healthy Urine. Normal urine has a yellowish color, a peculiar aromatic smell, an acid reaction, a specific gravity of 1015 to 1025. The amount in twenty-four hours is about 1500 cubic centimetres, or 50 ounces. On standing, a faint cloud, mostly of mucus, collects in the lower portion of the urine. On cooling, the urine may be slightly muddy, from the precipitation of urates, or, if the urine be alkaline, as it may be even in health, there may be a deposit of phosphates. Urine on standing and being exposed to the air, undergoes an acid fermentation, when it deposits a reddish precipitate, consisting largely of uric acid crystals. Later an alkaline fermentation sets in, the urine becoming paler and having a dirty gray sediment, and being cloudy from swarms of bacteria. At this time it has a strong ammoniacal odor. In hot weather, when the urine has been placed in an unclean vessel, especially when mucus, pus, or blood are present, the alkaline fermentation sets in early. In certain diseased conditions it may have commenced in the bladder.

THE QUANTITY OF URINE.

Normally a healthy adult of average size voids, in the course of twenty-four hours, 1500 cubic centimetres of urine. Within the limits of health, however, there may be great variations in the quantity of urine passed.

The amount will be increased by checking of perspiration, by the drinking of a large amount of fluid, as well as by certain diseased conditions, as, for instance, diabetes mellitus, diabetes insipidus, amyloid kidney, cirrhosis of the kidney. During convalescence from acute diseases, and when exudates or transudates are undergoing absorption, the amount of urine may be increased.

The amount of urine will be lessened when a small amount of fluid is taken, or when a large amount is lost through the skin or bowels. Thus in the hot weather of summer, when there is free perspiration, a patient may pass but half the normal amount of urine. In fevers, too, characterized by profuse sweating, the amount of urine is small. In cholera it may be suppressed. In some forms of nephritis, as acute nephritis, chronic parenchymatous nephritis, the urine may be diminished in amount. Nervous influences may change the amount of urine. In hysteria, for example, there may be complete suppression for a time, or, oftener, polyuria.

2. APPEARANCES OF THE URINE.

a. **Color.** The color of normal urine varies from a light yellow to a reddish yellow. The more concentrated the urine, the darker the color. With a large amount of water, as, for example, in diabetes, cirrhosis of the kidneys, hysteria, the urine is light colored. Febrile urine, the urine passed in the morning, the urine of congestion of the kidney, *e. g.*, during the course of valvular disease of the heart or emphysema, is darker colored. Blood in the urine changes the color, so that the urine becomes smoky or distinctly reddish. Hæmoglobin gives a reddish-brown tint. The bile pigments color the urine a yellowish-brown, to a brown or dark-green tint. Finely divided fatty matter, as in chyluria, causes the urine to be milky in appearance. Rarely the urine, especially upon standing, becomes very dark, or even black, when it contains melanin. Indican is regarded as the cause of the bluish or violet color of some urines (glaukuria). Certain foods and drugs may impart various tints to the urine. Senna and rhubarb cause the urine to be yellowish or brownish. Santonin colors it yellow, and if the urine be alkaline, red. Juniper gives a greenish-yellow tint. Turpentine, coffee, carbolic acid, tar, creosote, darken the urine.

b and *c.* **Clearness and Deposits.** The urine is usually clear and transparent. Upon cooling, it may be cloudy from the precipitation of urates. If the urine is alkaline, phosphates may be precipitated. The cloudy appearance may also be due to pus, to mucus, or epithelium. Decomposition of the urine before or after it has been voided, causes the urine to become cloudy from the swarms of bacteria.

3. ODOR.

Normal urine has an odor peculiar to itself. Concentrated and highly acid urine has a very penetrating odor. The odor of ammonia is clearly detected in urine that has undergone alkaline fermentation. Many drugs impart to the urine peculiar odors that sometimes alarm patients. Asafœtida, onions, radish, asparagus, change the odor of the urine in a marked degree. Turpentine gives to the urine the odor of violets.

4. SPECIFIC GRAVITY.

The specific gravity of the urine is normally about 1020. It varies, however, greatly even in health. With a large amount of water the specific gravity is low. Thus in hysteria, cirrhosis, after drinking, the specific gravity is found to be low. An exception to this rule is diabetes mellitus. With a small amount of water the specific gravity is high, as in fevers, acute nephritis, etc.

The urinometer with which the specific gravity is tested should be used at a temperature of about 60° F.; for every seven degrees above the temperature of 60° registers one degree less on the urinometer. When but a small quantity of urine is furnished for examination the amount is measured, and also the amount of distilled water it takes to fill the vessel to the height required to float the urinometer. We then multiply the number above 1000 that the instrument shows by the total number of volumes of the mixed fluid. This gives, approximately, the specific gravity.

The specific gravity depends on the solids contained in the urine. The total amount of solids is usually about 4 per cent.; that is, 60 to 70 grams in 1500 cubic centimetres, approximately 1000 grains. These figures are for an adult of average size and weight.

The total solids may be estimated, approximately, by multiplying the last two figures of the specific gravity by 2.33 (Hæser's coefficient). This gives the number of grams in each 1000 cubic centimetres of urine, or grains in each 1000 grains of urine, and from this the total solids in the total amount of urine can be calculated. For specific gravities below 1015 it is better to multiply by 2. To illustrate: If the amount be 1500 c.cm., specific gravity 1018, 18 × 2.33 = 41.94 = number grams in 1000 c.cm. In 1500 c.cm. there would be 1.5 times as much as in 1000 c.cm., or 41.94 × 1.5 = 62.91 grams.

If the amounts are expressed in fluidounces and grains instead of according to the decimal system, the following rule gives approximately the same results: Multiply the last two figures of the number representing the specific gravity by 1.1, and this product by the number of fluidounces. The result represents approximately the total solids in grains for twenty-four hours. Thus, let the amount for twenty-four hours be forty-eight ounces, specific gravity 1018, 18 × 1.1 = 19.8. 19.8 × 48 = 950.4 = number of grains of solids for twenty-four hours.

It is of great importance in many cases that the total solids should be estimated, as there is often a deficiency, though the amount of water is normal; or, *per contra*, with a small amount of urine, the amount of solids passed may be normal or even in excess. It is only safe to judge the specific gravity of urine from the twenty-four hours' specimen, as the diurnal variations in specific gravity are very great. In nephritis the sinking of the specific gravity, with the usual amount of urine, portends uræmia.

5. REACTION.

Normal urine is acid, because of the acid phosphates and urates. There are, however, great daily variations in the reaction, especially following meals, when the urine may be alkaline. In diseased conditions the acidity may be increased, as in rheumatism, gout, and lithiasis. Alkaline urine may be passed in certain depressed states of the general health, and in cases of retention and incontinence, from bladder or spinal disease of long standing. Urine that is ammoniacal when it is passed, always shows an unhealthy condition of the urinary tract, which permits of fermentation that is microbic in origin. The blue color imparted to red litmus by ammoniacal urine vanishes with heat. By noting the reaction of urine we may surmise the character of the deposits. Thus, urates or uric acid would be found in acid urine, while phosphates would be found as deposits in alkaline urine. "The persistence of pus in urine of acid reaction points to a renal origin.

Pus from the bladder, if persistent, usually renders the urine alkaline and ammoniacal." (Finlayson.)

Many drugs, as the salts of vegetable acids, as citrates, acetates, will cause the urine to become alkaline.

6. CHEMICAL EXAMINATION.

Organic Substances.

ALBUMIN.

" Whether albumin in any considerable amount can, under physiological conditions, be present in the urine is yet an open question." (Jaksch.) Besides the ordinary serum-albumin, that is found in the urine, globulin, peptone, albumose, oxyhæmoglobin, mucin, and fibrin are found.

Albuminuria.

Albuminuria means in its ordinarily accepted sense the passage of serum-albumin. This albumin may have its origin either in the kidney or in the urinary tract outside the kidneys.

Renal albuminuria is found as the result of (*a*) *disease of the kidneys*; (*b*) it may come from **circulatory disturbances** which influence the renal circulation, as in cases of heart disease. Here has also been classed the transitory albuminuria following epileptic attacks, cold baths, overexertion, etc. (*c*) **Febrile albuminuria** is well recognized, due possibly to altered blood pressure or to changes in the renal epithelium produced by bacteria or their products. (*d*) Albuminuria may appear in the course of **anæmia**, probably through alteration in the blood itself. (*e*) A class of cases exists where albuminuria is **intermittent** ~~or~~ cyclical, the exact cause not clearly understood. (*f*) Large amounts of albuminous food may cause albuminuria. From the above it can be seen that albuminuria *per se* is not a positive evidence of disease of the kidneys, certainly not of an inflammation of the kidney.

Accidental albuminuria signifies the passage in the urine of albumin, which has its origin outside the parenchyma of the kidney, as in the pelvis of the kidney, the ureter, the bladder, the urethra, or through abnormal communication with neighboring organs, as the lymph glands or vessels. Blood, pus, and semen may in this way give the tests for albumin.

Many tests for albumin have been proposed. The following are among the most reliable and easiest of application:

Test 1. Heller's Test. About one cubic centimetre of nitric acid is poured into a test-tube, and this is carefully overlaid by means of a pipette with about one cubic centimetre of urine. If albumin be present at the point of meeting of the acid and the urine, a white cloudy ring is seen. The test can also be applied by first placing the urine in the test-tube, holding the tube at an angle of 45 degrees and allowing the nitric acid to trickle slowly down the side of the tube to the bottom of the test-tube, to which point it sinks by virtue of its greater specific gravity. The rapidity with which the ring appears and its density furnish a rough quantitative test.

Test 2. The Heat Test. The urine, acidified by one or two drops of acetic acid, is heated in its upper stratum to the boiling point. If albumin be present it shows as a white cloud or as a distinct flocculent precipitate.

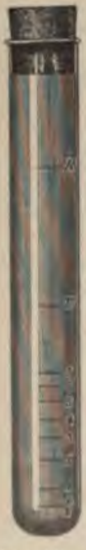
Test 3. Heat and Nitric Acid Test. The urine is boiled, and after boiling, nitric acid is added in small quantity, not more than $\frac{1}{15}$ of the volume of the urine that is employed. If albumin be present, it shows as a distinct cloud or precipitate. Phosphates that may have been precipitated by the heat are dissolved by the nitric acid. The cloud that was precipitated by the heat that does not dissolve on the addition of the acid is albumin. Care should be taken not to add too little or too much nitric acid.

Test 4. Acetic Acid and Ferrocyanide of Potassium Test. The urine is filtered, the filtrate rendered strongly acid with acetic acid, and a few drops of a 10 per cent. solution of ferrocyanide of potassium added. If albumin be present, a white cloud is seen, or, in case there is an abundance of albumin, a flocculent deposit.

Quantitative Tests. The amount of albumin can be estimated, approximately, by precipitating by heat and acetic acid and allowing the urine to stand for twelve or twenty-four hours. It can then be estimated as ten, twenty, fifty per cent. by volume. By Heller's test a rough estimate of the amount of albumin can be made by the rapidity with which the ring of albumin develops. The richer the urine in albumin, the more quickly the turbidity is developed. If in 100 cubic centimetres of urine there are 0.004 grams of albumin, the fluid begins to cloud after the lapse of forty seconds, and becomes plainly turbid in one and one-half minutes.

Esbach's Albuminimeter is a strong tube with its lower third graduated. When the percentage of albumin is to be determined, the tube is filled with urine to the mark U. The reagent (pure picric acid, 10 grams; pure citric acid, 20 grams; water, 970 cubic centimetres) is added until the fluid reaches the mark R. The urine and reagent are mixed by reversing and gently shaking the tube two or three times. The tube is closed with an India-rubber stopper, and allowed to stand upright for twenty-four hours. The height of the sediment is read off upon the scale. The numbers express the proportion of albumin in grams to the litre.

FIG. 52.



Esbach's Albuminimeter.

PEPTONE.

Biuret Test. Caustic potash is added to the urine, and a dilute solution of sulphate of copper is added drop by drop. In the presence of albumin the resulting peroxide of copper, a greenish precipitate, is dissolved, and the fluid becomes reddish violet. Peptone causes the fluid to become red. In case albumin is present in urine that is to be tested for peptone, it must first be removed. That is best done in the following manner: Ten c.cm. of a concentrated solution of acetate of soda and a few drops of perchloride of iron are added to 500 c.cm. of the urine until a red color remains. This is accurately neutralized with caustic potash, boiled, filtered, and allowed to cool. The filtrate should not disclose albumin upon the application of the heat and nitric acid test, nor upon the application of the biuret test.

A fairly satisfactory test for peptone is a saturated solution of tannic acid.

Peptonuria is divided by Jaksch into four classes: (1) Pyogenic peptonuria. Suppurative processes in the body are accompanied by the appearance of peptone in the urine. It is thus found in pneumonia in the stage of resolution, in suppurative pleurisy, purulent meningitis, etc. (2) Hæmatogenic, as in cases of scurvy. (3) Enterogenic, where peptone is absorbed directly from the intestines through ulcers. (4) Puerperal, during pregnancy and the puerperium.

The practical value of testing for peptonuria is occasionally seen where an attempt is made to establish the differential diagnosis between tubercular and suppurative meningitis. In the one case no peptone would be found, in the other, peptone would be present. Suppurative processes may be sometimes suspected when we detect peptone.

ALBUMOSE.

Albumose, formerly known as propetone, is now known to be a more complex substance, but cannot yet be said to be of clinical importance when it is detected in the urine.

GLOBULIN.

Globulin almost always occurs in conjunction with serum-albumin. It has the same clinical significance, therefore, as the latter substance. It can be detected in urine that also contains serum-albumin, by rendering the urine alkaline with ammonia, allowing it to stand for an hour, filtering, and adding to the filtrate its own volume of a saturated solution of sulphate of ammonia. A flocculent precipitate shows globulin.

FIBRIN.

Fibrin is found in the form of coagula when blood is present in the urine. To detect it, the clots should be separated by filtration, washed with water, and dissolved by boiling in a 1 per cent. solution of soda. The fluid is then tested by the ordinary tests for albumin.

BLOOD.

The presence of blood in the urine may be suspected from the color of the urine. The detection of the blood-corpuscles by the microscope is the crucial test.

Heller's test for blood consists in adding to the urine ¼ of its volume of liquor potassæ and boiling. The earthy phosphates are precipitated, and in the presence of blood the precipitate is colored a reddish-brown instead of a normal grayish-white.

The guaiac test for blood is made as follows: Add to the urine two cubic centimetres of tincture of guaiac and two cubic centimetres of old or ozonized turpentine, and shake thoroughly. In the presence of blood, after a short time, a blue color appears. Or the guaiac and turpentine may be shaken into an emulsion and an equal quantity of the suspected urine allowed to trickle down the side of the test-tube, inclined at an angle of 45°, and left to stand for a few moments. If blood be present, a bluish layer will appear between the urine below and the emulsion above.

Blood in the urine may come from the kidney, the ureter, the bladder, or the urethra. When from the *kidney*, it is intimately mixed with the urine, does not readily form a sediment, and clots are rarely found unless they are decolorized and of the shape of the calices or the ureter. Casts and renal epithelium are also found, and there are usually other evidences of renal disturbance, as pain, the presence of calculi, albumin, and so forth. Among the diseases of the kidney causing hæmaturia may be mentioned calculus, malignant growths, acute nephritis, tuberculosis, and trauma. The hemorrhagic diathesis, as in hæmophilia, scurvy, purpura, hemorrhagic smallpox, measles, etc., may produce hæmaturia, as also certain drugs, such as cantharides and turpentine.

Blood from the *bladder* is oftentimes in clots, less intimately mixed, and is prone to form an abundant sediment. There are usually presented evidences of vesical disturbance in frequent urination and pain over the region of the bladder. The first urine, too, is apt to be clear and the last bloody. Among the diseases of the bladder that might produce hæmaturia may be mentioned calculus, tumor, ulcer, tuberculosis, and inflammation.

Hæmoglobinuria has already been considered, in which the coloring matter is present in the urine; but no blood-corpuscles, or, at least, very few, and these imperfect, are found. Heller's test, or the guaiac test, shows the presence of blood pigment.

MUCIN.

Mucin in small quantities in the urine is not pathological. In larger quantities it may show catarrh of any portion of the urinary tract. It is often, in women, derived from the vagina. It can be detected by the addition of an excess of acetic acid, which renders the urine turbid. When nitric acid is added to the urine, as in testing for albumin, and allowed to trickle down to the bottom of the tube, a cloud of mucin is often seen about an inch above the line of union of the acid and the urine. If a large quantity of mucin is present when the urine settles it will form a visible gelatinous sediment at the bottom of the tube, so that no chemical test is necessary.

SUGAR.

Practically the only kind of sugar that is tested for in the urine is glucose. The test for grape sugar in the urine depends upon the following properties: (1) Grape sugar is colored brown when boiled with liquor potassæ. (2) On heating, grape sugar has the property of acting as a reducing agent. (3) With yeast, grape sugar is decomposed into alcohol and carbonic dioxide ($C_6H_{12}O_6=2C_2H_5OH+2CO_2$.)

1. **Moore's Test.** The urine is boiled with ⅓ its volume of liquor potassæ, and if grape sugar is present a brownish color appears.

2. **The Fermentation Test.** The fermentation test can be relied upon in all cases. A test-tube is filled with urine, inverted over a vessel containing the same fluid, and then a small bit of ordinary compressed yeast is placed in the tube. This is kept in a warm place, and if fermentation occurs with the formation of carbonic dioxide, which will accumulate in the upper part of the tube and gradually expel the urine, the presence of sugar is proven. Control-tubes, one with yeast and non-saccharine urine, and the other with urine to which no yeast has been added, can be employed if desired.

3. **Fehling's Test.** Fehling's solution consists of sulphate of copper, grains 90½; neutral tartrate of potassium, grains 364; solution of caustic soda, fluidounces 4; and distilled water to make up 6 ounces. A drachm of this solution is placed in the test-tube and boiled, and an equal quantity of urine added. When the mixture is heated, a reddish-yellow suboxide of copper is thrown down if sugar be present.

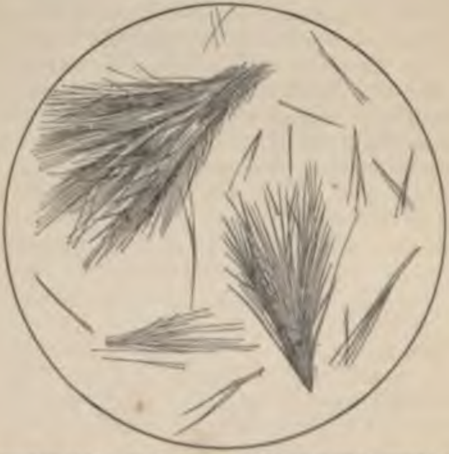
4. **Haines's Test.** Haines's test solution is a modification of Fehling's, and possesses this advantage, that it is not so apt to decompose with age. The method of preparation of Haines's solution is as follows: Take of pure sulphate of copper, 30 grains, and pure water, one-half fluidounce; make a perfect solution, and add pure glycerin ½ ounce; mix thoroughly, and add 5 ounces of liquor potassæ. A clear, transparent, stable, dark-blue liquid results. To make the test with Professor Haines's solution, about one drachm of the test solution is boiled, and should show no change. A few drops, not more than ten, of the suspected urine are added, and the mixture is again boiled. If sugar be present, a yellow or yellowish-red precipitate is thrown down. If no such precipitate appear, sugar is absent. The white flocculent deposit, when non-saccharine urine is added, consists of phosphates precipitated by the alkalies of the test liquid. These whitish phosphates should not be mistaken for the copper.

5. **Böttger's Test.** A concentrated solution of carbonate of soda is added to an equal quantity of urine, and a pinch of basic nitrate of bismuth is added. On boiling, if sugar be present, the bismuth turns black.

6. **Phenyl-hydrazin Test.** Two pinches ("twice as much as will lie on the point of a knife."—Jaksch) of phenyl-hydrazin-hydrochlorate and three of sodium acetate are dissolved

in about six c.cm. of urine in a test-tube, being well shaken. The tube is kept in boiling water for half an hour, and is afterward placed in cold water. If grape sugar is present a yellow deposit of phenyl-glucosazon is formed. If the deposit is very small it can be allowed to settle in a sediment glass, or in the centrifugal machine, and the sediment can be examined under the microscope, when beautiful, yellowish, feathery crystals are seen. If albumin be present it is better to rid the urine of the excess of albumin by boiling and filtering before applying this test. Better crystals, too, are obtained by gently heating, then filtering, then placing the test-tube for one hour in the boiling water, and allowing it to stand in this water until the water has gradually cooled.

FIG. 53.



Crystals of phenyl-glucosazon. (GIBSON and RUSSELL.)

Quantitative Estimation of Sugar.

The fermentation test gives the means of approximately estimating the quantity of the sugar in any given specimen of urine. The urine is allowed to ferment in a long-necked bottle, the opening being lightly covered to prevent evaporation. The specific gravity of the filtered urine is taken at the end of twenty-four hours, at the same temperature at which the fermentation has proceeded. The difference in the specific gravity before and after fermentation is then noted.

Each degree of difference corresponds to .219 per cent. of sugar. For example, urine which before the fermentation had a specific gravity of 1040, and after, a specific gravity of 1030, contains 2.19 per cent. of sugar.

By Fehling's solution an approximate quantitative estimation of the amount of sugar may be made. Two cubic centimetres of Fehling's solution are diluted with ten times their volume of water, and heated in a large test-tube. One to three drops of urine are then added, the whole again heated, observing whether the fluid still shows a blue color on holding it to the light. If there is still a blue color, a few more drops are added. It is again heated, again observed, and this process is repeated until the last trace of blue has completely disappeared, showing that all the cuprous oxide has been reduced. It is known that in the number of drops of urine required to decolorize there is exactly $\frac{1}{100}$ gram ($\frac{1}{6}$ of a grain) of sugar, and if we count twenty drops to one cubic centimetre we can calculate the percentage of sugar present. In order to save the time and trouble of making such a calculation at every examination, the following table will be found convenient (Seifert and Müller):

Drops.	Per ct. sugar.	Drops.	Per ct. sugar.	Drops.	Per ct. sugar.
1	20	10	2	25	0.8
2	10	11	1.8	30	0.6
3	6.6	12	1.6	40	0.5
4	5	13	1.5	50	0.4
5	4	14	1.4	60	0.3
6	3.3	15	1.3	70	0.28
7	2.8	16	1.2	80	0.25
8	2.5	18	1.1	90	0.21
9	2.2	20	1.0	100	0.20

BILE.

The bile pigments are found in the urine whenever there is an obstruction to the outflow of bile from the biliary passages. The bile, accumulating in the passages and in the liver, is absorbed by the veins and lymphatics, enters the circulation, and is eliminated by the kidneys. The detection of bile pigment in the urine does not in any manner show the character of the obstruction to the outflow of bile. When there is sufficient bile in the circulation the skin and other tissues are visibly stained, constituting jaundice. This is the hepatogenous jaundice. An alteration of the blood pigment within the bloodvessels, as occurs in pyæmia, may produce jaundice, which is known as hematogenous jaundice. Bile acids may be present in the urine, but are somewhat difficult of detection, and have no clinical value.

Bile pigment is ordinarily detected by the method of Gmelin. Nitric acid is allowed to trickle down the side of an inclined test-tube and settle to the bottom, as in testing for albumin with nitric acid. If bile be present at the line of union between the urine and the nitric acid, a colored ring, which ultimately becomes green (biliverdin), is seen. A modification of this test, which shows the colors very perfectly, is made by soaking clean, white, filter paper, which has been proven by a drop of nitric acid to be free from bile, with urine, and then placing upon this filter paper soaked with urine, a drop of nitric acid. About the drop is seen the greenish ring characteristic of bile pigments.

UROBILIN

is found in the urine in the case of fevers, in diseases where there is a destruction of blood cells, as in scurvy, and occasionally in Addison's disease. In diseases of the liver, such as cirrhosis, it has been repeatedly detected. In chloroform narcosis, in pernicious anæmia, in the hemorrhagic diathesis, in cases where blood has accumulated in different parts of the body, as after cerebral hemorrhage, hemorrhagic infarct, retro-uterine hæmatocele, urobilin has been detected. Urine containing this substance is usually dark. It has a yellowish foam upon it when shaken, as does the urine containing bile pigments.

The test for urobilin is made as follows: To the urine in a test-tube is added ammonia until the mixture is strongly alkaline. Eight to ten drops of a 10 per cent. solution of zinc chloride are added, and the mixture is filtered. In the presence of much urobilin the filtrate against a dark background is seen to be green, while held directly up to the light, of a glistening rose-red tint.

INDICAN.

Indican is a derivate of indol, which is the product of the decomposition of albuminous substances through the agency of putrefactive bacteria. It is found in the urine in conditions of health, especially if the diet be rich in meat. Increased putrefactive changes in the intestine, or in any other portion of the body, may produce indicanuria. Thus, a putrefactive peritonitis or pleuritis would produce it. It is found in abundance in cholera infantum. Large amounts of indican in the urine may be regarded as an evidence of the putrefaction of albuminous material in some portion of the body, though a simple, severe constipation may produce indicanuria.

The test for indican (Jaffé) is as follows: To a cubic centimetre of urine an equal volume of hydrochloric acid is added. Drop by drop there is then added, with shaking, a solution of some hypochlorite, *e. g.*, calcium hypochlorite, until a blue color appears. Too much hypochlorite hinders the formation of the indican. A few cubic centimetres of chloroform can then be added and the whole shaken, the chloroform becoming blue.

The second simple test is the following: Thirty cubic centimetres of urine are mixed with an equal quantity of hydrochloric acid, 1 to 3 drops of dilute nitric acid are added, and the solution boiled, giving it a dark color. After cooling it is shaken with ether. In the presence of indigo-blue, the liquid will be covered with a blue foam, while the ether itself becomes changed to a rose or violet color.

ACETONE.

Acetone is tested for by adding a few drops of freshly prepared nitro-ferrocyanide of sodium to the urine, and a strong solution of sodic hydrate until it is decidedly alkaline. When a yellowish or crimson tint appears, one to three drops of strong acetic acid are added, and if acetone be present a purplish tint is formed at the point of contact with the acetic acid and the mixture.

A more accurate test is made by distilling the urine with a little hydrochloric or phosphoric acid, and testing the distillate for acetone by *Lieben's test.* A few drops of a solution of iodine in iodide of potassium, and a few drops of potassic hydrate are added to a few cubic centimetres of the distillate. A yellowish-white precipitate of iodoform giving off the characteristic odor indicates acetone.

Acetonuria, to a limited extent, is physiological, especially if the food is rich in albumin. Pathological acetonuria is divided into the following forms (Jaksch): (1) Febrile; (2) diabetic; (3) accompanying certain forms of carcinoma which have not yet induced inanition; (4) inanition acetonuria; (5) in psychoses; (6) as an auto-intoxication; (7) in digestive disturbances.

Diacetic acid is found in some cases of diabetes and febrile processes, especially in children.

MELANINE.

Melanine is found in the urine in some cases of pigment-carcinoma, or develops by an oxidation process after the urine has stood. Such urine, on standing, becomes dark. Rarely, the coloring matter is found in masses of appreciable size. Melanine can be detected by the addition of bromine water, which produces a yellowish precipitate that gradually blackens.

LIPURIA.

Lipuria, or fat in the urine, may be present in some cases of chronic nephritis, phosphorus poisoning, diabetes mellitus, pyonephrosis, and chyluria. Physiologically, it may be observed during pregnancy. The urine is cloudy, or even slightly milky, the cloud vanishing upon shaking the urine with ether. Fatty acids are sometimes present in the urine. This condition is known as lipaciduria.

CHYLURIA.

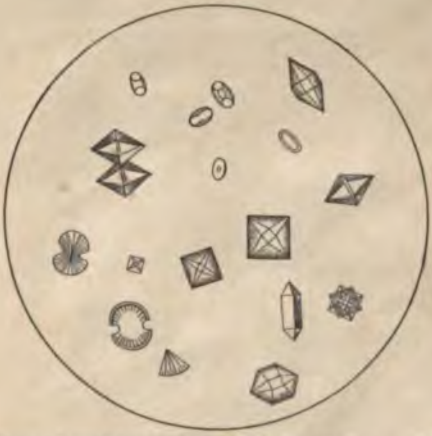
The urine in these cases resembles milk in color and has a creamy layer of fat that forms upon its surface. By shaking with ether, the greater portion of the fat is removed and the urine clears up. Hemorrhage may be associated with chyluria. The embryos of the filaria may be found in the urine of chyluric patients as well as in the blood, and especially during the night.

OXALURIA.

Oxalic acid, usually in combination with calcium, is a constituent of the normal urine. It may, however, appear in large amount, so that the characteristic octahedral or dumb-bell crystals are rapidly deposited when the urine settles. They can then be perceived as a deposit by the naked eye, but a microscopic examination is necessary for their identification. Where they are found in abundance in the urine a short time after it has been voided, their presence may be looked upon as pathological, as *oxaluria*. More accurate results are only obtained by a quantitative estimation of oxalic acid.

Oxaluria is often attended by a peculiar complexus of symptoms, so that it is sometimes described as a separate disease. Patients thus afflicted are usually those who are worn out by excessive work, especially brain work. Care and anxiety, overstudy, excessive sexual indulgence seem to be, at times, followed by this condition. The appetite is often deranged; there may be complaint of a dull pain in the loins. Mentally these patients are irritable, and frequently hypochondriacal. The urine is generally of high specific gravity, contains a large amount of urea, often a cloud of mucus, and a trace of albumin. The oxalates are often mixed with deposits of urates or uric acid.

FIG. 54.



Various forms of crystals of calcium oxalate. (GIBSON and RUSSELL.)

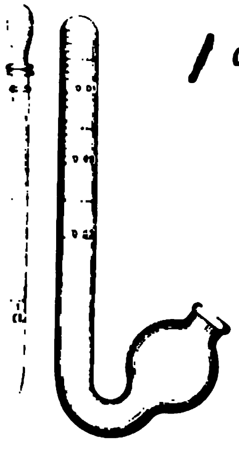
UREA.

From twenty to forty grams of urea are eliminated daily by the urine. This includes the greater part of the nitrogen taken with the food. Urea can be demonstrated by evaporating the urine to about half its bulk and adding an equal quantity of nitric acid. On cooling, shiny rhombic crystals of nitrate of urea are seen.

The quantitative estimation of urea is of considerable value, both as means of diagnosis and of prognosis. Urea is eliminated in increased amount in fever, diabetes, in phosphorus poisoning, in dyspnœa, and whenever there is an increase in the amount of albumin ingested. It is decreased where the diet contains a small amount of nitrogen; furthermore, in inanition, uræmia, and acute yellow atrophy of the liver.

The ordinary method of estimating the amount of urea is that by which the urea is decomposed by means of sodium hyprobromite and the volume of nitrogen measured, and from this the amount of urea estimated. Doremus's ureometer affords a cheap, simple, and reliable means of making this test. The apparatus consists (as shown in the cut) of a pipette and a graduated tube and bulb. The reagents necessary are: (*a*) solution of sodium hydrate, 100 grams to 250 cubic centimetres of water (six ounces to one pint of water), which should be kept in a bottle with a paraffined or rubber stopper; (*b*) bromine. Sodium hyprobromite is prepared by adding one cubic centimetre of bromine to ten cubic centimetres of the sodium hydrate solution, and diluting with ten cubic centimetres of water. Pour the sodium hydrate solution into the ureometer to the mark , then by means of the pipette add one cubic centimetre of bromine, and dilute with sufficient water to fill the long arm and bend. Tilt the ureometer and allow the hyprobromite to flow into the long arm and fill it completely. Draw the urine into the pipette to the graduation (1 c.cm.), pass the pipette into the ureometer as far as the bend, and compress the nipple *slowly*. The urine will

FIG. 55.



1 ℥ = 30 cc.

Doremus's ureometer.

rise through the hyprobromite, and the gas (nitrogen) evolved will collect in the upper part of the tube, and, after allowing the tube to stand for a few minutes, can be measured by the marks on the tube. Each division indicates 0.001 of a gram of urea in one cubic centimetre of urine. The quantity of urea voided in twenty-four hours is ascertained by multiplying the result of the test by the number of cubic centimetres of urine passed during that period. The weight of urea in 100 volumes of urine, (so-called percentage), is found by multiplying the result of the test by 100.

Thus, if a patient passes 1500 c.cm. of urine in twenty-four hours, and by the ureometer there is found to be nitrogen enough to fill the tubeto the nineteenth mark, there are 0.019 gram of urea in the one cubic centimetre of urine. In the total urine 0.019×1500=28.5 grams Or, to express the result in another way, there is 1.9 per cent. of urea (the normal amount being about 2 per cent.).

PTOMAINES AND TOXALBUMINS.

Certain toxic alkaloidal substances are found in minute traces even in normal urine. Under certain conditions these substances are found in greater abundance. While accurate methods for the detection of these bodies are still too uncertain or too complicated for practical clinical use, yet it is ot value to note the division into the various forms of toxæmia or toxicosis, as they have been classified by von Jaksch, who has given special attention to this important subject:

(*a*) **Retention toxicosis.** Clinical symptoms depending upon the retention of the physiological bases. Under this division is included uræmia.

b. **Noso-toxicosis.** Clinical symptoms referable to the presence of basic products which are formed in the system (*e. g.*, the blood), in disease, and eliminated with the urine.

c. **Auto-toxicosis.** Clinical symptoms caused by the formation of toxic basic substances, from morbid material, such as pathological fluids lodged in certain portions of the body. These bases are absorbed and then give rise to evidences of poisoning, *e. g.*, the symptoms following the absorption of putrid pus.

d. **Exogenic-torticosis.** Clinical symptoms induced by the action of toxic bases taken into the system with the food, *e. g.*, the poison of cheese and sausages.

7. MICROSCOPIC EXAMINATION OF THE URINE.

When the urine is to be examined microscopically it should always be allowed to settle, and a small portion of the sediment placed on the object glass by means of the pipette. For certain purposes, *e. g.*, the examination for casts, it may be necessary that the urine should stand for twenty-four or forty-eight hours, in order that the very light hyaline casts, whose specific gravity is nearly identical with that of the urine, should have time to settle. To prevent decomposition in the urine standing for such a length of time, some antiseptic, as a little boric acid, chloral, thymol, resorcin, may be added. This does not interfere with the examination of the sediment. It is usually best to take a large, rather than a small, drop of urine and to examine first with a low power, and then with the higher power. Care must be taken in examining such transparent substances as hyaline casts, that the light be not too strong. Rapid sedimentation can be accomplished by the use of any one of the many good centrifugal machines now on the market.

BLOOD.

The detection of blood by chemical means has already been referred to. The crucial test is always the microscopic or spectroscopic examination. Bloody urine is of a smoky or reddish tint, and usually has a sediment consisting of blood-corpuscles. Under the microscope these are seen of the same size and shape as the corpuscles in blood that is freshly drawn. Corpuscles from the kidney, however, are liable to be decolorized, so that but little is seen save a rim representing the contour of the red blood globule. To these washed-out rings the name *phantom corpuscles* has been applied.

LEUCOCYTES.

A few leucocytes may be found in the urine, and not be indicative of any diseased condition. In cases of inflammation of the kidney, or any portion of the urinary tract, leucocytes are found in great numbers, constituting oftentimes an abundant sediment of pus. These white blood-corpuscles are always polynuclear. They are usually unaltered in shape and appearance, the nuclei being distinctly visible. In alkaline urine, however, the corpuscles swell, the nuclei disappear, and the corpuscles become glossy and homogeneous. In cases where there is doubt as to the diagnosis between epithelium and altered white blood-corpuscles, a weak solution of methyl-blue can be used as a stain, which brings out the nuclei distinctly; or an iodo-potassic iodide solution, when the pus corpuscles become brown and the epithelium yellow. When the leucocytes have their origin in the ureter and pelvis the sediment is oftener flocculent, slimy, and translucent, and not so abundant as in cases of cystitis. In the latter disease there is usually a copious, thick, viscid deposit. When liquor potassæ is added freely to urine that contains pus, the solution becomes thick and viscid.

EPITHELIUM.

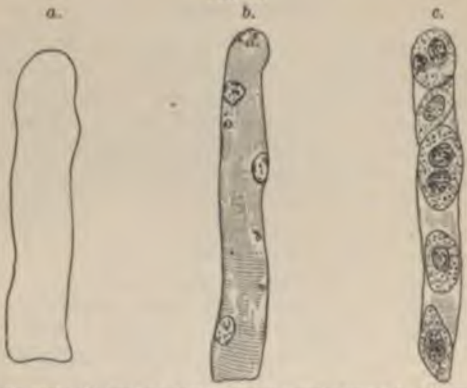
It is of importance to be able to recognize epithelium that comes from the kidney, as distinguished from that having its origin in the pelvis, in the ureter, the bladder, and the urethra or the vagina. The epithelium from the kidney usually has its cells smaller than those from the other portions of the urinary tract. The cells are polyhedral, finely granular, the nuclei of an oval shape and of large size. Often the epithelial cells are united one to another, and in conditions of acute inflammation of the kidney the cells lining the tubule may be shed *en masse*, constituting an epithelial cast. These cells may be seen singly or together, or upon hyaline casts. The cells may be in all stages of preservation, some appearing fresh and normal, others with swollen, indistinct nuclei, abundant granular matter, or oil globules, and evidence of degenerative processes in the kidney.

CASTS.

A cast, oftentimes called a tube-cast, is a mould of a renal tubule. While almost always indicative of renal disease, they have been occasionally found in urine that was non-albuminous and where there was no reason to believe that the kidney was diseased. Various divisions of casts have been made. They are divided by some into unorganized casts and organized casts. The casts of the first class are rarely found save in infants, and in some cases of gout and renal congestion. They consist of crystalline bodies, chiefly urates and hæmatoidin. The second group contains several varieties of casts, the exact origin of some of which is still a matter of theory. Organized casts may be made up of cells, as red blood-corpuscles, leucocytes, epithelial cells, or they may be made up of the products of cellular change, as the granular, waxy, fatty, and hyaline casts.

Blood Casts. Casts consisting of pure blood are not common. Such a cast is caused by a hemorrhage taking place into the tubule, from which the coagulated blood is extruded. Oftener the blood cast, so-called, is a hyaline cast upon which numerous red blood-corpuscles are carried. Blood casts are evidence of an acute inflammation of the kidney or of an acute exacerbation of a chronic nephritis. White blood-corpuscles have occasionally been found aggregated into the shape of the tubules. They, too, are evidence of an acute inflammation of the kidney.

FIG. 56.



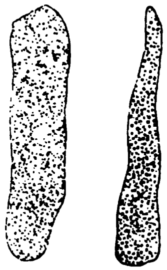
(*a*) Hyaline cast; (*b*) hyaline cast with leucocytes upon it; (*c*) hyaline cast covered with renal epithelium. (JAKSCH.)

Epithelial casts are, as their name implies, made up entirely of renal epithelium which has been shed from the uriniferous tubules. This wholesale desquamation takes place oftenest in acute nephritis. Epithelial cells are frequently seen clinging in numbers to a hyaline cast, and to such a cast the name *epithelial* is sometimes applied. These epithelial cells, it is to be remembered, whether in the form of a true cast or lodged upon a hyaline cast, are to be closely examined for evidences of change, such as atrophy, fatty degeneration, etc.

Bacteria are sometimes seen collected into the shape of a uriniferous tubule, and resemble very closely granular casts, but they resist the action of caustic potash and nitric acid. These casts, which are rarely found, are usually evidence of a septic pyelonephritis or of septic embolism of the kidney.

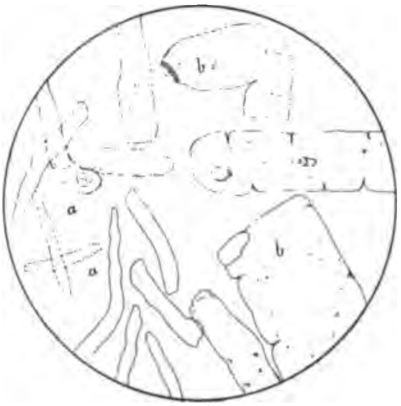
Granular casts usually consist of fine particles requiring a high power of the microscope for their recognition. On the other hand, the granules may be plainly made out because of their coarseness. These casts probably represent degenerate epithelial or blood casts, and are indicative of nephritis. They vary in color from a light yellow to a rather dark brown, and they vary also in breadth and length. They give evidence of an advanced degenerative process in the kidney.

FIG. 57.



Granular casts. (JAKSCH.)

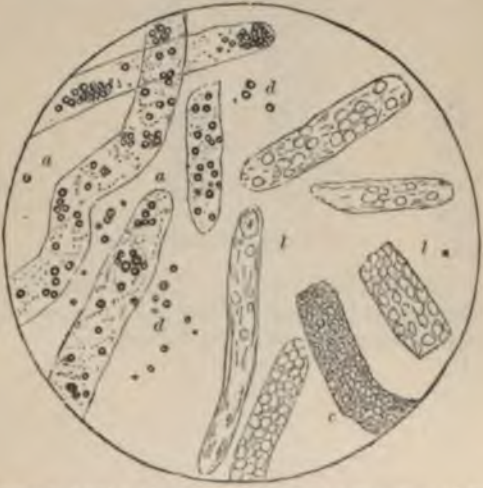
FIG. 58.



Hyaline and waxy casts. *a*. From a case of chronic Bright's disease of eight months' duration. *b*. From a case of chronic Bright's disease (large white kidney). *c*. From a case of chronic Bright's disease (contracted kidney with fatty degeneration). (ROBERTS.)

Waxy Casts. The casts that go by the name of waxy casts are broad, opaque, yellowish, oftentimes long, or, on the contrary, short as though broken into. They are not found exclusively in amyloid kidney, as was formerly taught, but may be found as well in acute and chronic nephritis. They may exhibit the amyloid reaction even in the absence of amyloid degeneration of the kidney.

FIG. 59.



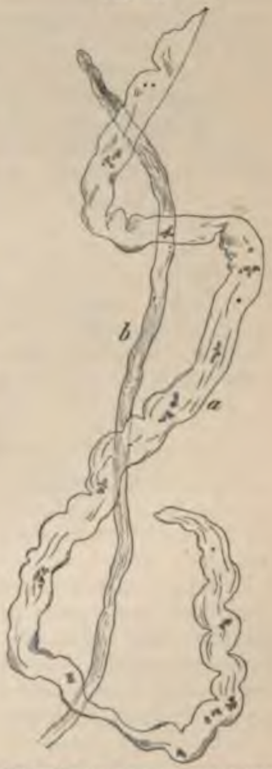
a. Fatty casts; *b* and *c*, blood casts; *d*, free fat molecules. (ROBERTS.)

Fatty Casts. Pure fatty casts are rarely found; that is to say, casts made up entirely of fatty globules or fatty crystals. Oftener the fat globules are seen upon granular or hyaline casts. They are of value as showing the tendency to fatty degeneration of the renal tissues.

Hyaline casts are found in the urine oftener than any other variety. They are pale or colorless, transparent casts, usually of a regular, oval outline, about two or three times as long as they are wide. In order to discover them, the light must not be too strong, and the most careful focussing is necessary, and at times a weak staining solution, as methyl-blue, can be used to advantage. These casts are often covered with fine, granular material, with epithelium, with oil globules, with red or white blood-corpuscles. To these hyaline casts covered with these various substances the names granular, epithelial, bloody, fatty, are often applied. In the strict sense, however, the terms should be limited to casts made up solely of granular material, of blood, of oil, epithelium, etc., and hyaline casts covered with these substances should be designated as hyaline casts with epithelium, hyaline casts with red blood corpuscles, etc. The detection of hyaline casts usually gives evidence of renal disease, yet they have been found in conditions where the previous and subsequent history has shown that no pathological state of the kidney existed, as, for instance, following an epileptic paroxysm. It is not always wise, therefore, upon the detection of hyaline casts alone, that is, without epithelium, leucocytes, granular matter, etc., to make a diagnosis of Bright's disease.

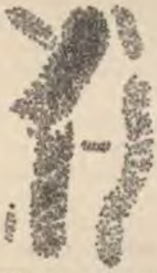
Another variety of cast, known as the *cylindroid,* is found chiefly in renal congestion, and especially in the urine of children. These bodies are long, ribbon-like, with edges slightly indented, the cylindroids being usually much curved upon themselves.

FIG. 60.



Cylindroids from a congested kidney. (JAKSCH.)

FIG. 61.



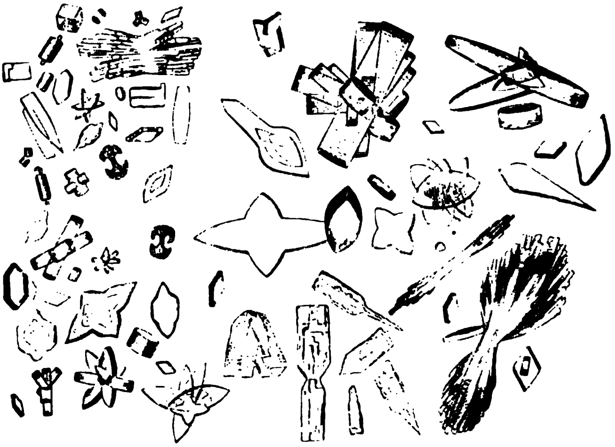
Casts consisting of urates. (JAKSCH.)

URIC ACID AND URATES.

Uric acid is a normal constituent of healthy urine, from one-half a gram to two grams being eliminated daily, the larger amount being excreted when an animal diet is taken. The kidney probably acts as an organ of excretion only, the uric acid being formed in the liver or in the muscles.

Uric acid is but slightly soluble, and when present in the urine, even when not in pathological quantity, is readily thrown down as a reddish deposit, showing under the microscope as reddish or yellowish-brown rhombs or prisms, spikes, and rods, frequently grouped in fan or sheaf-like bodies. The precipitation may be due to an excess of uric acid, or more often to excessive acidity of the urine.

FIG. 62.



Various forms of uric acid crystals. (FINLAYSON.)

To test a precipitate for uric acid add a drop of strong nitric acid, heat to dryness, and touch the reddish-brown deposit resulting on the white porcelain capsule, with a drop of a strong solution of ammonia. A crimson tint at once develops. If caustic potash be employed in place of ammonia a violet color is produced.

Uric acid is commonly found in combination with sodium and ammonium, as urates. These salts are met with in amorphous and crystalline forms. **Amorphous urates**, commonly a mixture of those of sodium, ammonium, potassium, calcium, are frequently seen as a fine, granular deposit of pinkish color, the tint depending on the amount of coloring matter in the urine. A fine film is also often seen upon the sides of the vessel containing the urine. Heat redissolves the urates. Their precipitation may be due to an excess in the urine, as in fevers and cases of altered metabolism, but may be entirely physiological and due to the cooling of the urine or to its excessive acidity.

Crystalline urates of sodium form a grayish-yellow deposit, consisting of irregular crystals with numerous spinous processes projecting from them. This deposit is rarely found save in very acid urine, as in gout and fevers. Precipitated within the urinary tract, it may produce irritation or lead to the formation of calculi. Urate of ammonium as a crystalline deposit is found during alkaline fermentation in combination with phosphates. In form, the crystals are opaque, spherical masses, or of dumb-bell shape.

Lithæmia, lithiasis, uric acid diathesis are terms used to designate a condition due to faulty metabolism, probably largely primarily hepatic in its origin, in which uric acid and urates are formed in excess and appear in the urine in abundance. The tendency to lithiasis is in some instances hereditary. Among the phenomena commonly attributed to this diathesis are gout, calculi, arterio sclerosis, with its consequences, such as aneurism, cerebral hemorrhage, cardiac hypertrophy, chronic interstitial nephritis, headaches, neuralgias, "bilious attacks," various skin eruptions, chronic bronchial catarrh, asthma, etc.

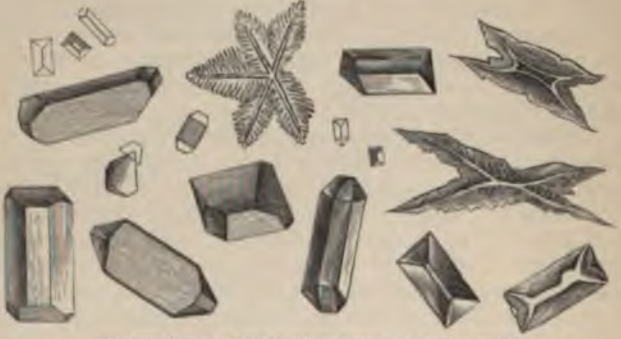
PHOSPHATES.

In the urine there are found phosphates of sodium, potassium, ammonium, calcium, and magnesium. Acid, neutral, and basic salts are formed. The neutral phosphates of the alkaline earths are but slightly soluble in urine, and their basic phosphates less so. When urine is boiled, the acid and neutral phosphates of the alkaline earths that may be held in solution, become converted into the insoluble basic salts, so that the urine becomes cloudy, and, on cooling, these phosphates show as a sediment. Phosphates occur as a deposit in alkaline or weakly acid urine. They are soluble in acetic acid.

The triple phosphate (ammonio-magnesium phosphate) shows under the microscope as crystals of large size, colorless, of coffin-lid shape, soluble in acetic acid. They are found in urine alkaline by virtue of ammonia, very rarely in acid urine. Feathery crystals and snow-flake forms may also be found.

Calcium phosphate occurs in alkaline urine or feebly acid urine that is becoming alkaline. The deposit may be made up of amorphous granules, or of acicular crystals, or of pointed wedge-shaped crystals sometimes grouped in clusters. From the urates and uric acid they are to be distinguished by the fact of their occurrence in urine that is alkaline or only feebly acid, and by their solubility in dilute acids, as acetic acid.

FIG. 63.



Various forms of triple phosphates. (FINLAYSON.)

Magnesium phosphate (basic) occurs in the form of large refracting, elongated, rhombic tablets. They are found in feebly acid or alkaline urine, dissolving by addition of acetic acid. Urine containing these earthy phosphates as a precipitate is often alkaline from an excess of alkaline bicarbonates, so that the addition of an acid causes brisk effervescence.

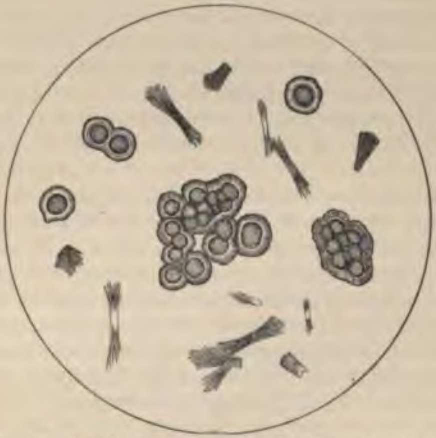
CHLORIDES.

The most important chloride in the urine is that of sodium. The amount of salt taken daily with the food may influence largely the amount of sodium chloride eliminated. Normally 10 to 15 grams are excreted in twenty-four hours. It is seldom necessary to test for chlorides for diagnostic purposes. In fevers, some gastric disorders, chronic nephritis, and particularly in acute croupous pneumonia, the chlorides are diminished. For the method of quantitative estimation some one of the larger works is to be consulted.

LEUCIN AND TYROSIN.

Leucin and tyrosin are found in acute yellow atrophy of the liver and in phosphorus poisoning. The former substance is seen under the microscope as yellow, glossy globules, often with radiating lines. Tyrosin is seen in the form of fine bundles of needles, or globules. Leucin and tyrosin are sometimes found as a sediment in urine, or it may be necessary, in order to prove their presence, to evaporate the urine to a syrupy consistence, allow it to cool, when the crystals formed can be detected by the microscope.

FIG. 64.



Crystals of leucin (spherules) and tyrosin (needles) from the urinary sediment of a case of acute yellow atrophy of the liver. (EICHHORST.)

INFLAMMATIONS OF THE KIDNEY.

GENERAL CONSIDERATIONS—SYMPTOMS COMMON TO ALL FORMS OF NEPHRITIS.

1. ALBUMINURIA.

There are few cases of Bright's disease in which, at some time, albumin is not present in the urine. In acute nephritis and chronic parenchymatous nephritis, albumin is almost constantly present in abundance. In the contracted kidney the amount may be slight, and for many hours, or even days, all the urine that is voided may fail to show even a trace of albumin.

It is important, when albumin is found in the urine, to determine whether or not we have to deal with a renal albuminuria or an accidental albuminuria, that is, an albuminuria where the albumin has its origin in the kidney, or without the kidney, as in the pelvis, the ureter, the bladder, or the urethra. This can only be determined by a careful consideration of the symptoms of the patient, and by the urinary findings. Thus, the presence of casts or of renal epithelium would be indicative of a renal origin for the albumin. The absence of these substances, the presence of pus, of bladder epithelium, together with the symptoms of cystitis, would argue in favor of a vesical origin for the albumin. Where albuminuria is due to the presence of blood, pus, semen, the amount of albumin should correspond with the amount of the substance in the urine giving rise to the test for albumin, that is, the larger the amount of pus the more pronounced the test for albumin, and *vice versâ.*

When we have proven that the albuminuria is renal, it is also necessary that we should determine whether the kidney trouble is primary or secondary. This has to be done largely upon the subjective and objective history of the patient. Among the causes of renal albuminuria, located outside the kidney, should be remembered cardiac disease, emphysema, the acute infectious diseases, severe anæmias, and the transitory albuminurias, such as follow epileptic attacks, the cyclical albuminuria, and that following overexertion.

2. CASTS, EPITHELIUM, ETC.

The significance of casts has been hinted at in connection with the description of their varieties. Casts in the urine are almost, without exception, indicative of organic disease of the kidney. The cases in which casts have appeared in the urine, and the kidney has been shown to have been healthy, are rare. The presence of degenerate epithelium, granular matter, and of oil globules, either grouped together as casts, or upon hyaline casts, or separately, indicates a severe or advanced stage of inflammation of the kidney. In the acute inflammations and in the exacerbations of chronic nephritis, red and white blood-corpuscles and true epithelial casts are more frequently seen. Hyaline casts may be present in all forms of Bright's disease, and the same may be said of the waxy casts, though the latter are found oftener in amyloid kidney than in any other form of renal disease. Some notion, too, as to the advancement of the disease may be gained by the breadth of these casts. Where casts are very wide it may be safely assumed that the tubule from which the cast originates has become widened by the destruction of the epithelium that lined it.

3. CHANGES IN THE CIRCULATORY APPARATUS.

It is very rare to find a case of chronic Bright's disease in which there are no changes in the heart or bloodvessels, or both. Without doubt, the heart may be primarily affected, leading to a secondary disease of the kidney, or the heart and kidneys may be affected in the general sclerotic change, as in cases of diffuse arterio-sclerosis. In other cases, the kidney is unquestionably primarily affected, and the heart undergoes a secondary hypertrophy with consequent increase in arterial tension. Various explanations have been offered for this phenomenon, but none absolutely satisfactory. The clinical fact, however, remains undisputed that in chronic interstitial nephritis there is always cardiac hypertrophy. In chronic parenchymatous nephritis there is almost always cardiac hypertrophy, and in acute nephritis that runs a lengthy course there are not infrequently cardiac changes. The theory which to-day has the most advocates is that advanced by Bright, that the retention in the blood of urinary constituents, chiefly urea, in some way produces the circulatory changes. The other theories are the mechanical theories, which tend to show that the obstruction to circulation in the kidneys from the diseased glomeruli must be followed by an increase of arterial pressure, in order to force the normal amount of arterial blood through the kidneys.

One should not forget, in any case of cardiac hypertrophy with increased arterial tension, to examine into the condition of the kidney, and particularly where there is no cardiac cause for the hypertrophy found. One should remember, too, that the increased tension does not necessarily mean arterio sclerosis. With arterio-sclerosis there is increased tension, but increased tension may exist without the marked sclerotic changes. The sclerosed radial artery is distinctly palpable when the current of blood is shut off by pressure between the point of palpation and the heart. A radial artery with normal walls, when emptied of its blood, cannot be felt so as to be distinguished from neighboring structures.

4. ŒDEMA.

The œdema of renal disease is regarded by some as due almost exclusively to the retention of water in the body, because of the failure of the diseased kidney to eliminate it. By others the exact opposite is believed, namely, that the œdema is primary, and the diminished elimination of water by the kidneys secondary. It is probable that in many cases some change in the vessel wall, by which it becomes more pervious and allows water to escape and to accumulate in the connective-tissue spaces is to be looked upon as the cause of the œdema. In many cases of nephritis, particularly of the chronic interstitial variety, the œdema is due to the same cause as the œdema of valvular disease of the heart, the heart in this form of nephritis, toward the last, becoming dilated and weakened, in a manner identical with that of valvular disease.

Dropsy may be an early symptom, as in acute nephritis and the chronic parenchymatous variety. It may be totally absent, as in cases of contracted kidney. In the chronic parenchymatous variety it may reach an extreme degree. Noticeable first in the eyelids and the dependent portions of the body, it gradually becomes universal, and the serous cavities are also filled with the transudate. In cases of nephritis, accompanied by great shortness of breath and cardiac palpitation, the chest cavities should always be examined for hydrothorax. Œdema of the lungs, œdema of the glottis or of the uvula, may be serious complications of any form of nephritis. The dropsical fluid resembles a watery blood serum. It is of low specific gravity and the amount of albumin in this fluid is very slight.

5. URÆMIA.

In diseases of the kidney there may be diminished elimination of water and of solid constituents of the urine. So little water may be passed that the solids, in normal amount, cannot be eliminated in solution. Such a condition is found in acute nephritis. On the contrary, a large amount of water may be passed and still the total amount of solids be deficient, as in chronic interstitial nephritis. When, from any cause, the solids that should be eliminated by the urine are retained in the system there is produced a condition to which the name uræmia has been given. While other substances may have a deleterious influence, and particularly certain alkaloidal compounds, that are normally excreted in small amount by the urine, it is probable that urea is the chief substance that causes the phenomena that are called uræmic. It was because of the early belief that urea was this poisonous substance that the name uræmia was applied to this condition. It is certainly true that in most cases of uræmia there is found a deficiency of urea, either because of the small amount of water that is passed, or because of the very small percentage of urea in the urine. The use of the ureometer is here of practical importance when we are attempting to recognize an impending uræmia. If the percentage of urea is found persistently small, uræmia is to be dreaded.

The symptoms of uræmia may come on suddenly in a very acute form, with no prodromata. The patient, in apparently good health, may be stricken down with a convulsion, or may pass rapidly into a comatose state. Not infrequently patients in uræmic coma are picked up in the street, supposedly intoxicated. Often, however, there are prodromal symptoms, such as persistent severe headache, dizziness or blindness, nausea, perhaps slight muscular twitchings.

The convulsion is often first indicated by the rolling upward of the eyeballs, followed soon by twitching of the muscles of the face, and general clonic convulsions of all the muscles of the body. The convulsion may last for a few seconds or for many seconds. The pupils are usually dilated, the face becomes purple, respiration seems almost absolutely suspended, the pulse is small and rapid. As the convulsion ceases a deep inspiration is taken, the color returns to the face, the pulse becomes fuller and stronger, and the patient breathes with a stertorous respiration. Consciousness may be lost for many minutes, or the patient may remain in a condition of stupor or coma, broken only by the advent of the second convulsion. Twenty or thirty attacks may occur within twenty-four hours. After each convulsion the patient is weaker than before. He may die during a convulsion or may become gradually exhausted and die in the interval between convulsions.

Among uræmic symptoms other than convulsion and coma are several phenomena that deserve attention. Pain, especially severe headache, is common. This headache may be very intense, and should always be looked upon as a suspicious circumstance in one known to have kidney disease or in a pregnant woman. Neuralgic pains in other parts of the body are not uncommon. Vomiting and diarrhœa may be uræmic. This vomiting usually occurs without any known dietetic error, is persistent and uncontrollable. Motor disturbances, especially slight twitchings, are occasionally seen without the occurrence of a general convulsion. Amaurosis may come on quite suddenly and may be complete. The pupils usually react to light, and a perfectly normal retina is seen with the ophthalmoscope. This amaurosis is probably of central origin. It usually disappears within the course of a few days. This is to be distinguished from the albuminuric retinitis to be spoken of later. The pulse of a patient suffering from chronic uræmia is often very slow, tense, and hard. At the time of the convulsion it becomes more rapid, as already described. During convulsion, and especially just preceding death, the temperature may rise to a great height, even to 106° or 108°. Uræmic asthma or uræmic dyspnœa may be the only evidence of uræmia. The patient is taken with an unaccountable dyspnœa, becomes cyanotic, pants rapidly for breath, and over the chest there are heard numerous, moist and dry râles, much as in asthmatic bronchitis. Uræmic symptoms are more apt to appear in the chronic interstitial nephritis than in any other form of Bright's disease.

These symptoms above enumerated are common to all forms of nephritis, and should be remembered by the student whenever he examines a patient with reference to disease of the kidney. I have found it of value to remember them by a little mnemonic device, namely, by the vowels of the alphabet.

A. Albumin.

E. Extraordinary substances in the urine (casts, epithelium, blood, etc.).

I. Increase of tension. (Circulatory changes.)

O. Œdema.

U. Uræmia.

NEPHRITIS.

Acute Nephritis.

Acute nephritis occurs most frequently during or immediately following acute infectious diseases, particularly diphtheria or scarlet fever. Exposure to cold, pregnancy, the use of certain irritating drugs that are eliminated through the kidneys, as turpentine, cantharides, chlorate of potassium, etc., may be the exciting causes.

There is at times a slight initial chill, with malaise and a moderate degree of fever. There may be pain in the lumbar region. Headache may come on early, and persist throughout the course of the disease. Nausea and vomiting are often troublesome from the start. In other cases the onset is much more insidious, and the first thing that is noticed is, perhaps, that in the morning, the face, especially the eyelids, is puffy and swollen, and that the countenance has a pallid look.

The diagnosis is made positively upon an examination of the urine. This is always diminished in amount. There may be in the course of twenty-four hours but one or two ounces passed, or even but a few drops. Complete anuria may exist for many hours. The urine is highly concentrated, of high specific gravity, loaded with albumin, and contains blood, often in the form of casts of the uriniferous tubules, renal epithelium that may be shed as a cast, and numerous hyaline casts. The blood may be present in such quantities that it is easily recognized by the reddish color that it imparts to the urine, and the abundant sediment that it forms. The microscopical examination is conclusive in that it reveals in greater abundance than in any other form of nephritis, epithelial casts, blood casts, and epithelium. The numerous hyaline casts are often covered with red and white blood-corpuscles and with epithelial cells.

The duration of acute nephritis varies. There may be for several days a moderate fever with an accompanying rapid pulse. The vomiting and nausea may continue, often accompanied by diarrhœa, and the dropsical manifestations may be marked. The patient becomes quite rapidly anæmic. In other cases, after a day or two, the amount of urine is increased, becoming even excessive, the fever diminishes, the pain disappears, the stomach and bowels again resume their normal functions. The œdema vanishes and the urine shows the albumin steadily growing less and less. These cases usually make a complete and perfect recovery. A slow and intermittent pulse is often noted during convalescence.

Uræmic symptoms may develop at any time during the course of acute nephritis, particularly when the amount of urine is very scarce. The uræmia may be manifested, apart from the vomiting and diarrhœa previously mentioned, by the most severe headache, tinnitus aurium, blurring of vision, great drowsiness, urinous odor of the breath, muscular cramps and twitchings, or by general uræmic convulsions that may be frequently repeated and terminate in coma.

During the course of chronic nephritis there may be acute exacerbations that differ in no respects, at least clinically, from acute nephritis. There is the same œdema, nausea, vomiting, headache, the same scanty high-colored urine, rich in albumin, blood, and epithelial casts. It should be remembered that the urinary sediment, in cases of acute nephritis of several days' duration, may show epithelium that has undergone degeneration; fat globules may be observed, and even large waxy casts are sometimes found.

The diagnosis rests almost exclusively upon the knowledge of the existence of the predisposing cause, such as scarlet fever, exposure to cold and wet, an alcoholic excess, upon the suddenness of the onset, the rapid development of œdema, and particularly upon the urinalysis. The scanty, smoky urine, of high specific gravity, loaded with albumin, with a heavy sediment, containing hyaline, bloody and epithelial casts, and free epithelial cells and blood-corpuscles, is found alone in acute nephritis or the exacerbations of the chronic form of Bright's disease.

Chronic Parenchymatous Nephritis.

In many cases there can be found no cause for this form of nephritis. It occurs oftener in males during adult life; oftener, too, in those who are the victims of alcoholism or syphilis, and who are exposed to cold or wet. Some cases, though not many, follow an attack of acute nephritis.

The symptoms manifest themselves rather slowly; oftentimes by those of a gradual, progressive anæmia, loss of strength, restlessness, headache, derangement of appetite. These slight disturbances may not alarm the patient, and especially as they progress so slowly; but when dropsy is noted, usually in the face upon rising in the morning, the physician is consulted.

He finds upon examination that not alone is there slight dropsy in the face, but that toward evening there is a little œdema about the ankles. The pulse shows an increase in arterial tension, the heart the evidences of left ventricular hypertrophy. The aortic second sound of the heart, in particular, is accentuated. On examining the urine, it is found to be scanty in amount, of normal appearance, but upon standing, throwing down quite an abundant sediment. The albumin is abundant, and the microscope reveals hyaline, granular, fatty casts, as well as epithelial cells, some fairly well preserved and others markedly degenerate. The specific gravity of the urine is usually rather high, from 1020 to 1030.

As the disease progresses the patient loses strength and flesh, though the loss of flesh is oftentimes not noticeable because of the increasing œdema. General anasarca is as pronounced in some cases of chronic parenchymatous nephritis as in any other disease.

Not alone may the dropsical transudate collect in the subcutaneous connective tissue, where swelling is visible to the naked eye, but as well in the serous cavities. Hydroperitoneum, hydropericardium, and particularly hydrothorax, are frequently noted, and it is of the greatest importance to recognize the latter condition, because of the danger attending the accumulation of a large amount of fluid in both pleural cavities. In all cases of Bright's disease where great dyspnœa is complained of, with difficulty in lying on one side at night, the chest should be most carefully examined for an accumulation of fluid. Uræmic symptoms may occur in this form of Bright's disease, often manifesting themselves as severe headaches, with vomiting, diarrhœa, temporary amaurosis. Convulsions and coma may likewise occur, although they are not as common as in the contracted kidney.

There can scarcely be any question as to the diagnosis, when the urine is carefully examined. The urine loaded with albumin, hyaline, fatty and granular casts, the urine diminished in amount, taken in connection with the marked œdema, the anæmia, the increased arterial tension, the cardiac hypertrophy, and the various manifestations of uræmia, make a picture presented by no other disease.

Chronic Interstitial Nephritis. Contracted Kidney.

The predisposing causes of this common affection are oftentimes very difficult to determine. Adult life and old age, the male sex, chronic lead-poisoning, chronic congestion of the kidney, as from heart disease, all favor or may produce this condition. So, too, the contracted kidney may be but one manifestation of a general sclerotic process in the bloodvessels, and any condition which favors this change, as syphilis, gout, the uric acid diathesis, favors the development of the contracted kidney.

No disease is more insidious in its onset than this. The patient may be a sufferer from chronic interstitial nephritis for years, but may show no symptom that attracts his attention, or that of any of his friends, to the fact that he is not a well man. There is usually a very gradual loss of strength, depending in a measure upon the slowly progressing form of anæmia. Disturbance of the appetite may be next noted, the appetite becoming somewhat irregular. Dyspnœa upon exertion, with palpitation of the heart, slight headache without any known cause, dizziness, insomnia, neuralgic pains are among the premonitory symptoms of this disease. Albuminuric retinitis may cause gradual or quite sudden dimness of vision.

An examination of the patient at this time will show that the heart has been for months gradually enlarging, until the physician finds the characteristic evidences of cardiac hypertrophy, the apex-beat displaced to the left and downward, diffuse, forcible, the second sound at the base accentuated, and the artery at the wrist showing a marked increase in tension. The artery is often, too, sclerotic. Even when the current of blood is shut off by pressure between the point of examination and the heart the artery can be distinctly felt by the finger, proving that its walls are thickened. Œdema in this form of Bright's disease may be totally absent. It is seldom found to any marked extent.

The urine of chronic interstitial nephritis, in typical cases, is increased in amount, from 60 to 100 ounces being passed in twenty-four hours. It is pale in color, of low specific gravity, seldom over 1012, has a very small amount of albumin, and a few hyaline and granular casts, with epithelium that may show granular matter and oil globules. The urine in these cases should always be collected for twenty-four hours, as the product of a single micturition may show nothing abnormal. The urine, even for one or more days at a time, may show no albumin and no casts, and it is said that in some cases of contracted kidney no albumin had been detected through life, though repeated examinations had been made.

In this form of nephritis uræmic symptoms may develop suddenly, often preceded by severe, blinding headaches and uncontrollable vomiting. The convulsions succeed and may be repeated at frequent intervals, so that twenty or thirty may occur during the course of twenty-four hours. Between these convulsions the patient is usually unconscious, and lapses into a condition of complete coma, in which he may die.

Among the complications of chronic interstitial nephritis should be mentioned retinitis, valvular disease of the heart, chiefly a relative incompetency of the mitral valves, cerebral hemorrhage (increased tension and the diseased vessel wall presenting the two conditions favorable for the rupture of bloodvessels), and inflammation of the serous membranes, as pleurisy, pericarditis, peritonitis.

The diagnosis of this form of Bright's disease is not so readily made as of the two preceding forms. The main factors in diagnosis are the large amount of urine, the small amount of albumin and casts, the marked cardiac and vascular changes, the slight degree of œdema, with the frequent occurrence of uræmic manifestations. In all cases of unexplained neuralgia this disease should be sought for. No case of cardiac hypertrophy without valvular disease, should escape without careful examination of the urine. So, too, where there are nausea and vomiting, diarrhœa without any dietetic error or gastro-intestinal disease to account for the occurrence, the kidneys should be carefully examined as the possible cause of the disturbance. Bronchitis and asthma are often found in patients with Bright's disease; and particularly should sudden, intense dyspnœa, with asthmatic symptoms, excite suspicion of the existence of contracted kidney. The anæmia of the disease makes it sometimes resemble the severer forms of essential anæmia. The existence, however, of cardiac hypertrophy, increased tension, casts in the urine, render the diagnosis comparatively easy.

Amyloid Kidney.

Amyloid kidney occurs as a consequence of chronic suppurative processes, particularly in the bones, of tuberculosis, especially when associated with suppuration, and syphilis. It is extremely rare for the kidney alone to be affected, usually the liver, spleen, and intestine sharing in the amyloid change. The symptoms of involvement of the kidney may be almost or quite masked by those of the primary disease, and there may be associated with the amyloid degeneration a chronic inflammation of the kidney, so that it is extremely difficult to tell how many of the symptoms referable to the kidney are due to the one condition, and how many to the other.

The diagnosis rests upon the examination of the urine, the finding of the liver and spleen enlarged, and the discovery of some cause, such as suppuration, tuberculosis, or syphilis, that might induce the change. The urine, while usually increased in amount, may be normal. It is commonly pale, contains albumin, hyaline, granular and waxy casts. These casts will occasionally show an amyloid reaction under the microscope. It is said to be quite characteristic of amyloid kidney that there is great variation from day to day in the amount of albumin found. The diagnosis without a knowledge of the primary disease, without the proof of accompanying enlargement of the liver and spleen, can scarcely be made with certainty, though the disease may be suspected when the amount of urine is increased and it contains a very large amount of albumin, this amount varying greatly from day to day, and when, too, there are no cardiac changes. Œdema may or may not be present.

TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN THE VARIOUS FORMS OF BRIGHT'S DISEASE.

	Acute nephritis.	Chronic parenchymatous nephritis.	Chronic interstitial nephritis	Amyloid kidney.
Albuminuria	Constant and marked.	Constant and marked.	Slight and often intermittent.	Marked, though with great variation.
Extraordinary substances, (casts, etc.)	Blood; hyaline, epithelial casts; epithelium in abundance.	Hyaline, granular, fatty casts, numerous.	Hyaline, granular casts, few.	Hyaline, granular, waxy casts, usually abundant.
Increased tension; vascular changes.	Slight change, unless disease protracted.	Usually some hypertrophy and increased tension.	Hypertrophy and increased tension very marked.	Seldom hypertrophy, or increased tension.
Œdema . . .	Of sudden appearance, fairly well marked.	Excessive.	Slight or absent.	Often slight, or absent till late in the disease.
Uræmia . . .	May be convulsions or coma.	May be convulsions or coma, though not very common.	Very often convulsions and coma.	Seldom uræmic symptoms.
Water. Characteristics of urine.	Small amount, high specific gravity, smoky or bloody in color.	Small amount, high specific gravity.	Large amount, pale, low specific gravity.	Variable.

Suppurative Nephritis, Perinephritis, and Pyelitis.

Purulent inflammation of the kidney, of its pelvis, and of the tissues roundabout the kidney, present many symptoms in common. In all these diseased conditions there is usually some primary disease to which the renal difficulty is secondary. In purulent nephritis there is generally some acute infectious disease, or a malignant endocarditis as the primary affection, the infection being by way of the blood current, or the kidney structure becomes involved in suppurative inflammation, by extension of the suppurative inflammation of the pelvis into the uriniferous tubules. Perinephritis usually arises through traumatism, or as the result of suppurative inflammation in the immediate neighborhood of the perirenal connective tissue, as, for example, purulent nephritis or pyelitis, hepatic, psoas, or peri-appendicular abscess. Tubercular and actinomycotic abscesses may also be found in the perinephric region. A mild form of inflammation of the pelvis of the kidney is often found accompanying the acute infectious diseases, as typhus and smallpox. Irritating drugs, as cantharides and copaiba, may also produce pyelitis, and foreign bodies, as renal calculi or parasites, are often the exciting cause. The severer and suppurative forms of pyelitis come on, almost always, as an extension of the inflammation from below upward. Thus, a urethritis by extension, becomes a cystitis, ureteritis, and pyelitis.

In all these forms of inflammation there are first the symptoms of the primary disease, which may so overshadow the symptoms of the renal trouble as almost completely to mask the disease of the kidney.

The symptoms of suppuration-fever, sweating, chills or chilliness, emaciation, are present in all cases, but in varying degrees. Locally there is usually pain. This may be slight, may be present only upon movement of the body, or upon pressure, or may be of extreme severity. In a perinephritic abscess there is oftentimes seen a bulging in the lumbar region, over which the skin is reddened or œdematous, the swelling being painful upon pressure. An exploratory puncture gives pus. Purulent nephritis is usually a post-mortem rather than a clinical finding. The pain in the lumbar region, a trace of albumin in the urine, with possibly an increased number of leucocytes, if one of the minute abscesses ruptures into the uriniferous tubules, may be the only facts that point in the direction of a purulent nephritis.

Pyelitis is to be suspected when it is known that the causative factor is present, as a cystitis or a renal calculus, and where mucus and pus are discharged with the urine. Great difficulty oftentimes exists in determining whether the pyuria is due to a cystitis, a pyelitis, or both. An examination of the epithelial cells found in the sediment is rarely conclusive, that of the pelvis of the kidney and of the bladder being quite similar. The pelvic epithelium is usually found in the shape of long-tailed, triangular cells with a distinct nucleus, the cells oftentimes laid over one another as shingles, yet quite similar forms may be found from the wall of the bladder. Blood may be found in pyelitis where a calculus is in the pelvis.

The urine in pyelitis is usually acid; in cystitis usually alkaline. There is pain in the region of the kidney in pyelitis; pain in the hypogastric region in cystitis. Oftentimes the kidneys themselves become secondarily involved in inflammatory action where there is primary pyelitis. To this condition the name pyelonephritis is given. In these cases casts and albumin would be found in the urine. Where it is suspected that one ureter is discharging pus, the ureters, particularly in women, may be catheterized and the nature of the urine from either kidney determined.

Congestion of the Kidney.

Congestion of the kidney occurs oftenest in connection with cardiac disease or with emphysema. Clinically, the condition is only recognized by the alteration in the urine. The urine is decreased in amount, of high specific gravity, dark in color, contains an abundance of urates and of uric acid. Frequently albumin is found in slight or moderate amount. Hyaline casts, red and white blood-corpuscles are also present. The long continuance of renal congestion leads to permanent changes in the kidney, so that later a contracted kidney may result. It is characteristic of the urine in congestion of the kidney to approach more nearly the normal as the blood pressure becomes more nearly normal. This is a valuable point in diagnosis.

EMBOLIC INFARCTION OF THE KIDNEYS.

This condition is rarely of clinical importance except as one of the phenomena occurring during the course of valvular disease of the heart. An embolus lodging in one of the larger renal vessels usually produces sudden, sharp pain, referred to the region of the kidney, sometimes with slight symptoms of shock or collapse, and hæmaturia. The amount of blood is usually slight. Smaller infarctions occur without any symptoms. If, then, during the course of valvular disease there is sudden pain in the region of the kidney, followed by hæmaturia, renal infarct can be suspected.

TUMORS OF THE KIDNEY.

Carcinoma and sarcoma of the kidney may be primary in this organ. Sarcoma, except it be congenital, is, however, rare as a primary tumor of the kidney. Renal carcinoma is found oftenest at the two extremes of life. One kidney is usually affected, though double renal carcinomata have been found. The kidney may be changed to a large tumor weighing several pounds. By direct extension and by metastasis, other organs, as the liver or lungs, may be involved.

Pain is a common symptom of renal cancer; the tumor is almost always perceptible. This is felt in the lumbar region, and extends upward and inward. It is hard, may be smooth or rough, is dull upon percussion, though a loop of intestine, particularly the descending colon upon the left side, may lie between the tumor and the abdominal wall, so that a resonant note is heard. Neighboring organs, as the liver or the spleen, may be displaced by the enlarged kidney. The renal tumor does not move with respiration. The urinary examination may be negative. The healthy kidney may perform its function normally, and, by compensation, secrete the usual amount of healthy urine. Hæmaturia is, however, so commonly found in renal cancer as to be of great value in diagnosis. There are found, of course, the general symptoms of cancer, which symptoms, however, may appear quite late in the course of the disease. As in cases of malignant growth in any organ, the patient becomes weak, emaciated, and anæmic. The pulse in renal carcinoma is often strikingly rapid.

The location of the tumor with its position behind the large intestine, its failure to move with respiration, the hæmaturia, the cachexia, usually enable one to make a diagnosis of renal tumor, and this is especially the case if such conditions obtain in children. A tumor of the kidney may, however, be confused with ovarian tumors, psoas abscess, tumors of the spleen, or other enlargement of the kidney, as hydronephrosis, pyonephrosis, and echinococcus.

MOVABLE KIDNEY.

The movable kidney is found oftenest in women who have borne children, or in those who, because of poor general nutrition, have all of the tissues in a relaxed condition. The right kidney more frequently shows this peculiarity than the left. In many cases movable kidney causes no symptoms whatever, or but slight pains and feelings of distress in the abdomen. In other cases the pains are described as dragging, or even as colicky, and are severe. They may be referred to almost any part of the abdomen, though usually to the sacral and lumbar regions. Nausea and vomiting may be present, all these symptoms being aggravated by the movement of the patient, as by walking or jumping. The pains are oftentimes aggravated, too, when the patient lies upon the side opposite the affected one. Severer attacks may resemble attacks of renal colic; there may be sudden pain, chilliness, and vomiting, and more or less shock. The urine is diminished for many hours or a few days. It is probable that these severer symptoms are due to a twisting of the ureter, producing a temporary acute hydronephrosis.

Various nervous symptoms, in part reflex, in part, perhaps, hysterical, are found in patients the possessors of a floating kidney. Among these symptoms may be mentioned headache, gastric irritability, hypochondriasis, and even some of the graver manifestations of hysteria. Pressure of the kidney upon neighboring organs may produce various effects, as constipation, jaundice from pressure on the bile ducts, œdema of the lower extremities from pressure on the vena cava. Dilatation of the stomach has been found in connection with floating kidney. The exact relation between these two conditions has not been established. The kidney in patients with relaxed walls can oftentimes be felt as a smooth mass of the peculiar shape of the kidney, having at its hilum a pulsating vessel, the renal artery. It may even be grasped between the fingers and slipped back into its place. Pressure upon the kidney gives rise to a peculiar sickening sensation. Palpation of the kidney should always be attempted by the bimanual method, one hand being in the lumbar region, the other over the abdomen.

HYDRONEPHROSIS.

An obstruction of the ureter, as by an impacted calculus, by a stricture the result of inflammation, by pressure from without, as from new growths, causes the urine poured into the pelvis of the kidney to accumulate, gradually distend the pelvis, and produce the condition known as hydronephrosis. Pressure atrophy of the renal tissue will occur later, so that we may have the kidney merely one large sac, containing urine and the secretion of the mucous glands.

The diagnosis of the disease depends in a measure upon a knowledge of the primary disease, which causes the obstruction in the ureter. Usually there is a distinct swelling that gradually enlarges toward the hypochondrium and the median line, and may finally fill the entire abdomen. Fluctuation may be noted. The swelling does not move with respiration; it is dull upon percussion. Whenever the obstruction is not complete and is relieved at times, there may be diminution in the size of the mass coincident with a free discharge of urine. Exploratory puncture will reveal fluid containing urinary constituents, chiefly urea. In women, and recently in men, the ureters have been catheterized, proving that one ureter is dry.

The symptoms of this condition may be few, though usually there is pain in the region of the tumor, and various pressure symptoms. The amount of urine may be markedly diminished until compensatory hypertrophy of the other kidney has been established. Uræmic symptoms may develop.

In case pyogenic organisms are present, pus may be found within the kidney sac, when the condition is termed pyonephrosis. Here, in addition to the other findings just recorded, there would be the evidences of suppuration, fever, chills, sweats, emaciation, and exploratory puncture would reveal pus.

The diagnosis of this condition is easy when it is known that there is some cause existing for the occlusion of the ureter, otherwise the hydronephrosis may be very easily confused with other tumors of the kidney, with ovarian tumors, with tumors of the spleen, the liver, and of the gall-bladder. The diagnosis in these cases is to be made largely by exclusion or by the employment of the exploring needle.

NEPHROLITHIASIS.

Urinary concretions forming in the renal pelvis when very small are spoken of as renal sand, when a little larger, like gravel, as renal gravel, and when still larger as renal calculi. The renal calculus is usually about the size of a pea or bean, so that it can pass through the ureter. Calculi in the pelvis of the kidney may be much larger and may even fill the pelvis. Oftenest the calculus is of a brownish or blackish color, laminated, smooth, and made up of uric acid. The mulberry calculus, with a rough surface of a dark-brown color and very hard consistence, is made up of the oxalate of calcium. Softer stones made up of the phosphates are less often found in the kidney than in the bladder.

Renal calculus is found chiefly in those who secrete an acid urine. In some cases the excessive use of sour wines, drinking water that contains lime, a large amount of meat, seem to predispose to the formation of calculi. Uric acid stones are not infrequently found in gouty patients. A calculus in the pelvis of the kidney may not cause any pathological change in the pelvis, or it may, especially when its surface is rough, produce irritation and inflammation, lighting up a pyelitis. Simple catarrhal, diphtheritic, or purulent pyelitis may be the result of a renal calculus, and among the remoter consequences may be noticed pyelonephritis, or, from rupture, perinephritis. When a calculus obstructs the opening of the ureter or becomes impacted in the ureter, hydronephrosis results.

In the passage of a calculus from the pelvis of the kidney to the bladder, symptoms are produced to which the name renal colic has been given. Preceding the attack of colic there may have been symptoms pointing in the direction of a renal calculus. Renal gravel or sand may have been passed. Slight pain in the kidney may have been complained of. Hæmaturia may have been noticed, and if pyelitis or pyelonephritis has been set up, the symptoms of these conditions already enumerated would be present. Renal colic may come on without any exciting cause. Sometimes a sudden jarring of the body, as in jumping or riding, seems to excite it. The pain is extremely severe, is referred to the region of the kidney, from which it radiates along the line of the ureters down to the testicles and the penis. There may be retraction of the testicle upon the affected side. Nausea and vomiting of a reflex character are common, and the patient, if pain be very severe, may be thrown into a state of collapse, with feeble pulse, countenance indicative of suffering, subnormal temperature, and even syncope. During the attack small amounts of urine may be frequently passed that may contain blood from the pelvis of the kidney or from the lacerated ureter. Renal colic may last for a few hours or for several days, ceasing with the dropping of the stone into the bladder.

The diagnosis of nephrolithiasis is made upon the history of renal colic, the detection of the stone or of gravel in the urine, and upon periodical attacks of hæmaturia of renal origin. Renal cancer, renal tuberculosis, and parasites of the kidney, as well as the hemorrhagic diathesis, must be excluded as causes of hæmaturia. Where pyelitis is present, pelvic epithelium, pus, and mucus may be constantly present in the urine, and the picture will be varied according to the severity of the inflammation of the pelvis and of the kidney.

A condition not infrequently mistaken at first for renal colic, where the right kidney is involved, is hepatic colic; but in gall-stone colic the pain starts in front, just below the edge of the ribs, and radiates to the epigastric region and upward toward the right scapula and the right shoulder. In renal colic the pain starts posteriorly, radiates downward to the testicle and the penis. In hepatic colic there is usually tenderness on pressure over the region of the gall-bladder. There is no history of blood in the urine, no stone or gravel is passed in the urine, but there may be a history of previous similar attacks followed by jaundice, and the examination of the feces may show a gall-stone. Jaundice subsequent to an attack of colic is indicative of its hepatic origin.

Perforative appendicitis may, at the beginning of the attack, be diagnosed as renal colic; but a consideration of the points of origin of the pain, the rise in temperature, the tenderness in the right iliac region, the signs of local peritonitis, and the absence of urinary symptoms should enable one to make the diagnosis.

ECHINOCOCCUS OF THE KIDNEY.

Echinococcus cyst of the kidney is rarely bilateral. Symptoms may be lacking, the first evidence being afforded by the detection of a tumor. This is usually round, not very painful upon pressure, and may give a peculiar hydatid thrill or fremitus upon palpation. Whenever the sac bursts into the pelvis of the kidney, single cysts or hooklets may be passed in the urine, often with the symptoms of renal colic. In doubtful cases an exploratory puncture is justifiable, and here the microscopical examination of the fluid withdrawn will disclose the hooklets. Echinococcus cyst is to be differentiated from hydronephrosis, pyonephrosis, and in women from ovarian cyst.

TUBERCULOSIS OF THE GENITO-URINARY APPARATUS.

Secondary involvement of the genito-urinary apparatus in tubercular processes, as in miliary tuberculosis, is not uncommon. Primarily any portion of the genito-urinary tract may be attacked. This gives rise to a local tuberculosis, as of the kidney, the testicle, or the prostate gland, which may by direct extension and dissemination, involve a greater area of the genito-urinary tract.

The symptoms of genito-urinary tuberculosis are usually those of a chronic pyelo-cystitis. Pain in the region of the kidney, even colicky in character, may be noticed where the kidney is involved. The urine contains an abundant sediment that is seen under the microscope to consist of pus corpuscles, detritus, and, in almost every case, the tubercle bacillus. To examine for tubercle bacilli, several drops of urinary sediment are allowed to evaporate on a cover-slip, are then fixed and stained as in the method of staining the sputum for bacilli. Its detection in the sediment is a positive indication of involvement of some portion of the genito-urinary tract. Blood is not infrequently found in this form of tuberculosis. Seldom can the kidney be found enlarged by palpation, though occasionally this is true. Fever of a hectic character, with the constitutional evidences of tuberculosis, emaciation, weakness, anæmia, etc., are present. When the disease involves the bladder there is usually pain over the bladder. The urine is passed frequently, it is alkaline, contains an abundant sediment, and contains the tubercle bacillus. The prostate, the testicles, should always be examined in suspected cases of genito-urinary tuberculosis, because they may be primarily or secondarily involved, and they, unlike the kidney, are accessible to direct examination. Spermatorrhœa should excite suspicion of tuberculosis of the prostate and seminal vesicles. Cystoscopic examination should be practised in suspected and doubtful cases of tuberculosis of the bladder.

In advanced cases recovery does not occur. Death may occur from general weakness, from involvement of other organs, as the lungs, from amyloid disease, or with symptoms resembling somewhat the symptoms of pyelocystitis.

CYSTITIS.

The bladder is usually inflamed as the result of the introduction of organisms through the ureter. The unclean catheter and a gonorrhœal inflammation of the ureter are the two common ways in which cystitis arises. Certain drugs, as cantharides, in their elimination by the urine may cause inflammation of the bladder, and in cases of paralysis of the bladder, with incontinence of urine, bacteria gain entrance, so that in many nervous affections, among the terminal complications must be mentioned cystitis. Foreign bodies introduced through the urethra or formed within the bladder, as vesical calculi, may be the exciting cause of cystitis.

The symptoms may be slight or extremely severe. Following infection of the bladder with an unclean catheter there may be chills, quite marked fever, severe pain in the region of the bladder. The urine is passed frequently, with much pain, in small amounts. Vesical tenesmus is marked. The urine is slightly acid or even alkaline, contains an abundance of vesical epithelium, blood corpuscles, as well as the micrococcus ureæ. Through the influence of this organism, urea becomes changed into ammonium carbonate, frequently rendering the urine ammoniacal even before it is voided. In severe forms of cystitis entire shreds of necrotic tissue may be found in the urine. Blood corpuscles may be discovered by the microscope, or the hemorrhage may be so extensive as to cause blood-clots, visible to the naked eye, to be passed. Whenever there is pus and mucus in the urine there is albumin. This substance will vary according to the amount of mucus and pus. The greater the amount of pus, the greater the amount of albumin. The slimy threads in the urine, the "Tripper-Faden," are found in cystitis of gonorrhœal origin. Where the alkaline fermentation is very marked, symptoms that have been designated as ammoniæmia are sometimes present. It is supposed that ammonia is absorbed from the bladder, and produces the headache, vertigo, stupor, and nausea that characterize this condition. Chronic cases are characterized by frequent urination attended with some pain, tenderness over the bladder, and the presence of pus, mucus, and vesical epithelium in the urine.

NEW GROWTHS.

New growths may be found in the bladder, usually secondarily. The commonest is the papilloma. Hemorrhage is found in the case of this tumor; the blood-clots are often long and worm-like in appearance. An examination of the bladder with the cystoscope, with the sound, and per rectum, is of great value in enabling the diagnosis of this condition to be made.

Primary carcinoma of the bladder is rare. It usually causes the walls to be greatly thickened, so that the thickened walls may be palpated through the abdomen. Pain in the region of the bladder, frequent urination, may lead to the diagnosis of cystitis. Blood and pus may be found in the urine. Suspicion of the malignant character of the growth should always be excited whenever the patient is seen to lose flesh, become anæmic, and develop the cancerous cachexia. Secondary deposits should be looked for in all doubtful cases. The enlargement of the inguinal glands would point in the direction of a malignant growth.

DISEASES OF THE NERVOUS SYSTEM.

THE PERIPHERAL NERVES.

DISEASES OF THE SENSORY NERVES.

DISTURBANCE OF SENSIBILITY.

Absence of sensibility is known as anæsthesia; increased sensibility as hyperæsthesia; and certain abnormal sensations in the skin, such as prickling, tingling, numbness, formication, as paræsthesia. There are various varieties of cutaneous sensibility, among which may be mentioned tactile sensibility, the sense of locality, of pressure, of temperature, of pain, and the electro cutaneous sensibility. To these may be added also the muscular sense or muscular sensibility. Disturbances of sensibility may involve all of these varieties, or there may be partial paralysis of sensation. Thus the sense of pain may be diminished or absent, while the pressure sense remains intact.

In examining patients for alterations in the normal sensations it is necessary to have the patient's eyes closed and to examine symmetrical portions of the body. The examination should also be conducted with as little discomfort to the patient as possible, and as rapidly as possible, as many of these nervous, perhaps hysterical, patients are rendered more nervous and easily tired by a long, tedious examination, and they are apt also to become disgusted with the examination where it is protracted, and where they do not understand the meaning of all the different manœuvres of the physician.

Tactile sensibility, that is, the sensibility of the skin to simple contact without pressure and without the production of pain, is tested by drawing lightly over the surface of the skin some substance, as soft wool, cotton, a camel's-hair brush. Normally this light contact should be detected. Normally, also, the patient should be able to distinguish form and certain outward peculiarities of objects by the sense of touch. He should be able to distinguish, for instance, between a smooth and a rough object; he should be able to recognize well-known objects, such as a knife, scissors, key, coins.

Sense of Locality. Sense of locality is tested by having the patient tell what spot upon the body is touched by the examining finger or by the point of a pencil. Where the sense of locality is acute, the patient is able to tell when two points are brought simultaneously in contact with the skin, and to recognize the fact that there are two points. Normally this sense differs in different portions of the body. Thus, at the tip of the tongue two points are recognized as such at a distance of less than two millimetres apart; on the backs of the fingers, at a distance of two to three millimetres; on the cheeks, at a distance of eleven to fifteen millimetres; at the tip of the nose, twenty-two millimetres; on the forearm, forty millimetres; on the back, about sixty millimetres; on the leg, forty millimetres etc. In abnormal conditions the patient may not be able to recognize at these distances that two points are brought in contact with the skin. He may detect only one. In other cases, where one point is brought in contact, there is the sensation as though two points were present. To this peculiar symptom the term *polyæsthesia* has been applied. The instrument by which the sense of locality is usually tested is the æsthesiometer, a variety of tactile compass.

Sense of Pressure. A rough method of testing the sense of pressure, and one that is ordinarily serviceable, is by making pressure with different degrees of strength with the two hands upon corresponding parts of the body, and asking the patient on which side he feels the greater pressure. More accurate tests are made by supporting the part to be examined and placing coins or weights upon this part. Ordinarily an increase of pressure of about one-twentieth of the original pressure may be perceived. In conditions of disease, however, the pressure may be doubled without the patient perceiving that there is any increase.

Sense of Temperature. The temperature-sense is tested by filling two test-tubes, the one with hot, the other with cold water, and applying them successively to the skin, and noting whether the patient can tell when the hot and when the cold tube is in contact with the surface of the body. When the temperature-sense is lost, a hot or cold body may produce only a sensation of touch or of pressure, but not of temperature, or, in some cases there may be a perversion of the temperature-sense, and the patient may feel a cold body as a hot one, or *vice versâ.*

Sensations of pain are recognized by the patient when the point of a pin is touched to the skin, or when the skin is pinched. In abnormal conditions the sensation may be entirely absent, or there may be a retention of only the tactile sense. To this latter condition, that is, retention of tactile sense with loss of sense of pain, the name *analgesia* is given. It is important to make the distinction between pain-sense and tactile sense, as in some diseases, *e. g.*, syringomyelia, the diagnosis may hinge upon this fact. The *electro-cutaneous sensibility* is occasionally tested for, but is rarely necessary, as the results are practically those obtained by testing for sensibility of touch and of pain.

The muscular sense, which probably includes as well what might be termed the sensibility of the joint surfaces, ligaments, and tendons, is that sense by which the position of the different portions of the body is recognized by the patient. This can be tested by having the patient close the eyes, then putting the limbs in certain positions or through certain motions. We ask the patient to describe the position or the motion, or to place the corresponding limb in the same position, or to repeat the motions that have been passively performed for him by the examiner.

In certain pathological conditions, particularly spinal cord diseases, as locomotor ataxia, there may be variations from the normal sensations, to which the name *paræsthesia* has been given. These patients complain of the crawling of insects over the skin (formication), numbness, tingling, and prickling. With paræsthesia there is no outward irritant, or the sensation does not correspond to the irritant as when the ataxic feels as though he were walking on velvet, when perhaps he walks upon the bare floor. There are also abnormal *after-sensations*, as when the patient after the skin has been pinched, complains of a sense of heat or of numbness. The sensation is also in certain cases, as locomotor ataxia, delayed in its conduction to the perceptive centres. Following the prick of a pin upon the lower extremity of an ataxic, there may be a perceptible interval of many seconds, even fifteen or twenty seconds, before the pain is felt by the patient.

ANÆSTHESIA

may be peripheral, spinal, or cerebral; peripheral where the fault is in the conducting power of the nerve; spinal where the pathological condition is in the cord; cerebral where the perceptive centres fail to act.

Among the causes of anæsthesia of peripheral origin may be mentioned a chilling of the skin, the corrosive action of acids and alkalies, and of such drugs as cocaine, morphine, and belladonna. In this case the anæsthesia is due to an alteration in the *terminal sensory organs.* The *nerve-trunks* may also fail to conduct the sensation because of traumatism, compression, inflammation, or degeneration.

The *spinal cord* may fail to conduct the sensory impulse to the brain in a variety of diseases, as in locomotor ataxia, and compression of the cord. Remembering that the sensory fibres pass directly into the posterior gray horns, some, however, first entering the posterior columns—the "root zones"—we can understand why in certain spinal diseases, *e. g.*, tabes, pain is prominent, while in others, *e. g.*, anterior poliomyelitis, where the posterior horns and posterior columns are unaffected, pain is absent. The crossing over of the sensory fibres to the opposite side of the cord is not to be forgotten, particularly when we attempt to explain the phenomenon of such a disease as Brown-Séquard's paralysis.

Cerebral anæsthesia is frequently seen following hemorrhage. This is usually unilateral. If the hemorrhage involves the posterior portion of the internal capsule, half of the body opposite the side of the brain lesion will be anæsthetic, a condition known as hemianæsthesia. The action of certain anæsthetics and narcotics, as chloroform, morphine, etc., is due to their action upon the nerve cells of the brain. Functional or psychic anæsthesia is a term used to describe the loss of sensibility where no organic cause is found to account for the abnormal condition, but where psychic causes, as in hysteria, seem to be operative.

Anæsthesia is usually readily recognized, the patient complaining of numbness or entire loss of sensation in some portion of the body. Hysterical anæsthesia is, however, often overlooked by the patient. Anæsthesia is frequently accompanied by motor disturbances, often by paralysis, as well as by trophic changes. Where a portion of the body is anæsthetic—such as the fingers, the cornea, or the feet—which is in constant and almost involuntary use, there is a great alteration in the manner in which ordinary acts are performed. Especially is this true of the finer movements. Thus, patients stumble as they walk; they are unable to button the clothing rapidly, to hold a pin, to thread a needle. The anæsthesia of the cornea permits the lodging of foreign bodies upon the eye, without the patient's winking to remove them.

It is of great importance for the diagnosis of nervous diseases, as well as for the definite localization of pathological lesions, to note whether or not the anæsthesia is distributed over an area governed by some central area, by some set of nerves, or by some particular nerve. Thus, anæsthesia of one half of the body, hemianæsthesia, speaks for a unilateral cerebral lesion; anæsthesia of the lower segment of the body, paranæsthesia, for a bilateral lesion of the cord cutting off sensory impressions. Anæsthesia in the area of distribution of a single nerve usually signifies a lesion in the nerve itself or at its point of central origin. Irregular patches of anæsthesia not corresponding to the distribution of any one nerve, apparently lawless, may give aid in the recognition of hysteria.

DISTURBANCES OF THE MOTOR NERVES.

PARALYSIS.

By paralysis is meant the inability to move a muscle. The term is usually restricted to the voluntary muscles. Paralysis may be due to a lesion of the brain, of the spinal cord, of the nerve, or of the muscle itself; so that we may speak of a cerebral, a spinal, a peripheral, or a muscular paralysis. The lesion producing paralysis may be located anywhere in the course of the motor tract. This tract is the motor centre of the cerebral cortex, the internal capsule, the crus, the anterior half of the pons, the pyramids, the anterior surface of the medulla, the lateral pyramidal tract of the cord (most of the fibres decussate in the medulla, passing to the opposite lateral tract, a few passing down in the anterior pyramidal tract), the anterior gray columns, the ganglion cells, the anterior spinal roots, the peripheral nerves. A lesion located anywhere in the course of this tract will serve to shut off the motor impulse from the cerebral centres and will produce paralysis.

Paresis signifies partial or incomplete paralysis, a weakness of the muscles. By **hemiplegia** is meant a paralysis of half the body. This is the chief form of cerebral paralysis. **Monoplegia** signifies the paralysis of a single muscle or group of muscles. This is mainly due to a lesion of the cerebral cortex or of the peripheral nerve. By **paraplegia** is meant the paralysis of the two halves of the body below a certain segment of the cord. The term, as commonly employed, signifies a paralysis of the lower extremities. By brachial paraplegia is meant a paralysis of all parts of the body below the cervical segment. Paraplegia is almost always of spinal origin.

A lesion destroying the ganglion cells of the anterior cornua, or located between the ganglion cells and the muscle, will be attended by atrophy as well as by paralysis, so that the paralysis is spoken of as an **atrophic paralysis.** Attending it there is degenerative atrophy of both nerve and muscle. **Pseudo-hypertrophic paralysis** is that form in which the muscles, increased in volume through hypertrophy of a few fibres, but chiefly through the development of the interstitial fatty and connective tissue, are yet weak and paralytic, because the majority of the muscular fibres have undergone atrophy. If there is loss of motility that is due solely to atrophy, the condition is spoken of as a **simply atrophy,** not as paralysis. If, however, the paralysis is greater than the atrophy alone produces, the condition is known as **amyotrophic paralysis.** A distinction is often made between spastic and flaccid paralysis. A **spastic paralysis** is where, with a weakened condition of the muscles, the muscles are rigid, and exaggerated reflexes are found. The opposite condition, where the muscles are flabby, is known as **flaccid paralysis.**

The test for paralysis is the inability of the patient to move a voluntary muscle. In children and in those who are unconscious, the position in which the limb is held may give evidences of paralysis, or a failure of the muscles to respond to ordinary stimuli may reveal the fact. Where the muscle is but partially paralyzed, we test the degree of a paralysis both by the rapidity with which the muscle is moved, by the steadiness or the tremulousness of the movement, but chiefly by the resistance that is offered to the hand attempting to prevent the action of the muscle. True paralysis should be carefully distinguished from the immobility of muscles due to contractures, to anchylosis, and to pain.

Etiology of Paralysis. Paralysis may be due to functional or psychic causes, as in hysteria. Among the organic causes may be mentioned inflammations and degenerations, tumors, hemorrhages, emboli, thrombi, mechanical pressure as from aneurisms, from dislocated vertebræ. Toxic influences, as in cases of lead poisoning, the long-continued use of arsenic and copper, may induce paralysis. The ptomaines present in the blood in the acute infectious diseases, as, for example, diphtheria, typhoid fever, smallpox, dysentery, may produce paralysis. Cold, so-called rheumatism, may produce paralysis, usually of peripheral origin. And in a certain class of cases where abdominal organs are involved there are paralyses whose origin is not clearly understood, known as reflex paralyses.

VARIETIES OF ABNORMAL MOVEMENT.

By **spasm** is meant an involuntary movement of a muscle. While the cause is often unknown, it is assumed to be some irritation of the motor tract.

1. A **clonic spasm** is a short, coarse spasm, usually quite rapidly repeated, such as is seen in an epileptic convulsion.
2. A **tonic spasm** is one in which the muscle is rigidly contracted for a considerable length of time, as in trismus, tetanus, opisthotonos.
3. A **convulsion** is a spasm of numerous muscles or groups of muscles of an extremity, or of the entire body, typically seen in epilepsy, where the spasms are clonic or clonic-tonic.
4. **Rhythmical contractions** in single muscle groups, perfectly regular, are occasionally seen in cerebral hemorrhage and multiple sclerosis.
5. **Tremor** is a rapid, rhythmical movement of groups of muscles, the excursions being slight, and the muscular movements always producing movement in some joint. This is seen in paralysis agitans, in exophthalmic goitre, in alcoholism. In some cases, as, for instance, in paralysis agitans, voluntary effort will check the tremor for a time. In others, as in multiple sclerosis, an attempt at voluntary movement increases the tremor (intention tremor). Tremors are described as coarse, fine, slow, or rapid. They are often best seen, as, for instance, in alcoholics, by having the patient hold out the arm, extend the hand, and separate the fingers.
6. There are **single, slow contractions** or twitchings that are sometimes present in cord disease and are not clearly understood.

7. **Fibrillary muscular contractions** of muscular fibres or bundles of the fibres. These are wormlike movements only seen on close inspection, and met with in atrophied muscles. They are arrhythmic. They are seen in the tongue in glosso-labial paralysis, in the small muscles of the hand in progressive muscular atrophy.

8. **Choreic movements.** These are irregular, lawless movements, continuous, or with pauses, involving the whole body, or at times only one side or a single limb.

9. **Athetoid movements** are slow, almost ceaseless, usually met with in the arms and hands, though they may be found in the head and trunk. The fingers are extended, spread apart, flexed, moved over one another in an extremely odd and characteristic manner. Athetosis is a consequence of the cerebral palsy of children, though at times, in the absence of any known cause, it has been looked upon as a primary disease.

10. **Co-ordinated spasms.** These are forced movements, requiring the co-ordinated action of many groups of muscles, as, for example, circular movements, propulsive movements of paralysis agitans; also spasmodic laughing, hiccoughing, crying.

11. **Cataleptic rigidity,** where the muscles remain involuntarily in any position in which they are passively placed. It is chiefly seen in hysteria and in the hypnotized.

12. **Associated movements,** where, if the attempt is made to move one leg, the other moves also, or where a leg and an arm may move together.

13. **Cramps.** These are painful, tonic spasms, as, for example, in the calves of the legs in cases of arsenic posioning.

14. **Ataxia.** A break in the centripetal or centrifugal tract may produce ataxia. Abnormal muscular sense, sense of pressure, tactile sense, joint sense, may remove many of the controlling influences that render muscular action regular and orderly, so that it may become irregular and disorderly. Or with normal sensibility, motor impulses may be partly cut off from one muscle, or from a group of muscles, by some lesion in the motor tract, and the intended movement may be in part feebly or imperfectly performed, or two groups of muscles may not act synchronously as they should. This may result in ataxia. Ataxia is tested by having the patient touch the nose with the tip of the finger with the eyes closed; by having him carry a glass of water to the lips; by having him touch the knee of one leg with the heel of the opposite leg. It is often noted by the patient himself that in writing, or in but-

toning the clothing, the movements are uncertain and irregular. The gait of the ataxic where the ataxia is at all well marked is quite characteristic. He walks with a wide base, lifts the foot high, swings it out, and brings it down with a stamp. The gait is uncertain and unsteady, the patient keeps the eyes on the feet. With ataxia, the patient frequently, when the eyes are closed, sways and will fall to the ground unless caught. Ataxia and chorea, resembling somewhat one another, are readily distinguished when it is remembered that ataxia is noticed with motion; chorea with rest. The choreic makes first one kind of movement, and then another, with the arm or with the leg; the ataxic always makes the same kind of a false move.

THE REFLEXES.

By reflexes are meant involuntary movements that regularly appear on the application to some point on the body of a certain or definite stimulus. They are divided into superficial or cutaneous reflexes and the deep reflexes, the latter practically limited to the tendon reflexes.

Of the *cutaneous reflexes*, there are three that are of practical importance: 1. The abdominal reflex, whose seat is in the lower dorsal cord. A light stroke with the finger over the skin of the abdomen causes a contraction of the abdominal muscles of the corresponding side. 2. The cremasteric reflex. Pressure about three inches above the internal condyle of the femur, or an irritation of the skin on the inner side of the thigh, by the drawing of the finger nail or of the point of a pencil over it, produces a contraction of the cremasteric muscle that lifts the testicle on the corresponding side. 3. Tickling of the soles of the feet causes dorsal flexion of the toes, or even a withdrawal of the foot, and the bending of the knee and of the hip.

The Tendon Reflexes. The most important of the tendon reflexes is the *patellar tendon reflex*, or the knee phenomenon. The patient sits with the legs crossed, the muscles relaxed, and the attention directed away from the leg by his looking at the ceiling. The physician gives a light, sharp tap with the finger or the percussion hammer on the patellar tendon, and in response the quadriceps extensor contracts and the leg is extended with a jerk. In case the reflex is not manifested in this way, reinforcement, as it is termed, may be employed. The patient locks the fingers and draws with the hands with all his might, looking meanwhile toward the ceiling. This reinforces the tendon reflex. When a patient is bedridden, the reflex can be elicited by the physician lifting the leg by placing his left arm in the popliteal space; then when the muscles are relaxed the tendon can be percussed and the reflex elicited.

Among other reflexes that are occasionally of value are the triceps tendon reflex, elicited by percussing the triceps tendon, with the elbow bent; the periosteal reflex of the radius and of the ulna, percussion being made over the lower end of these bones, with the arm slightly bent; the masseter reflex and the ankle-clonus. The latter, which is present in cases of lateral sclerosis, is made manifest by a sudden passive extension of the foot when the leg is straight and the muscles relaxed; several clonic contractions of the muscles follow and the foot is thrown into alternate extension and flexion. What are known as idio-muscular contractions are sometimes tested for by direct mechanical stimulation of the muscle, as by a sudden stroke upon the muscle.

VASOMOTOR AND TROPHIC DISTURBANCES.

While the exact pathology of the vasomotor and trophic disturbances as they are met with in nervous diseases is not clearly understood, they should always be observed, as clinically they are often great aids to diagnosis. Vasomotor disturbances are shown by the warmth or coolness of the skin, by its pallor (vasomotor constriction), by the blue or reddish blush of the skin (vasomotor dilatation). The nervous influences may also increase or diminish glandular secretions, producing dryness of the mouth, salivation, anidrosis, hyperidrosis, etc. An œdema of nervous origin—the angioneurotic œdema—may appear. So, too, urticarial lesions, vitiligo, and other pigment anomalies may be present and due to vasomotor influences. It is questionable whether the pigmentation of Addison's disease is not due to the influence of the sympathetic system. Trophic disturbances are often present in disease of the cord or of the brain. Bedsores readily develop; there may be glossiness of the skin, dryness of the nails, loss of the nails or of the hair, and many changes in the bones and joints that must be looked upon as due to alteration of trophic influence (Charcot joint).

DISORDERS OF THE VASOMOTOR AND TROPHIC NERVES.

RAYNAUD'S DISEASE.

This disorder usually manifests itself suddenly, often after exposure to cold, in a pallor and numbness of the fingers, the hands, or the tips of the ears (stage of syncope). Following this pallor, which may last for a few minutes or for several hours, there is a stagnation of the capillary circulation and the fingers become livid and painful (stage of asphyxia). Later there may follow necrosis, as of the tips of the fingers, the edge of the ear (stage of local or symmetrical gangrene).

Accompanying the paroxysms of this peculiar disease there may be chill, cerebral and general nervous disturbances, hæmoglobinuria.

ANGIO-NEUROTIC ŒDEMA.

In this affection, "giant urticaria," there is a local, suddenly developing œdema, often attended by colicky pains, sometimes by nausea, vomiting. The œdema is transient. The lips, eyelids, fingers, hands, genitalia, legs, throat, and larynx may be attacked. Sudden œdema of the larynx may be fatal. An hereditary tendency can often be made out.

FACIAL HEMIATROPHY.

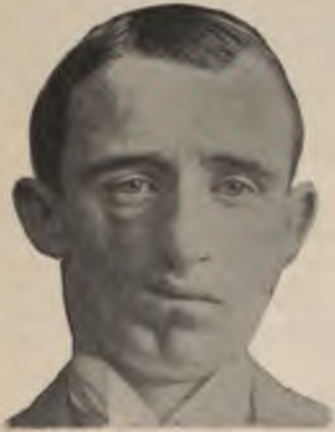
This usually begins as a wasting of the subcutaneous tissues of one side of the face. Gradually the muscles and the bone atrophy, the teeth fall out, and the hair is lost. The shrunken, wasted half of the face in such marked contrast to the healthy side, make it an affection usually recognized at a glance.

ACROMEGALY (Large Extremities).

In acromegaly there is an enlargement chiefly of the bones of the face and extremities. The lower jaw increases in size until the lower teeth may project beyond the upper. The nostrils, eyelids, and ears may also show hypertrophy. The hands, fingers, and wrists increase in size, the hands looking coarse and spade-like. The feet likewise become larger.

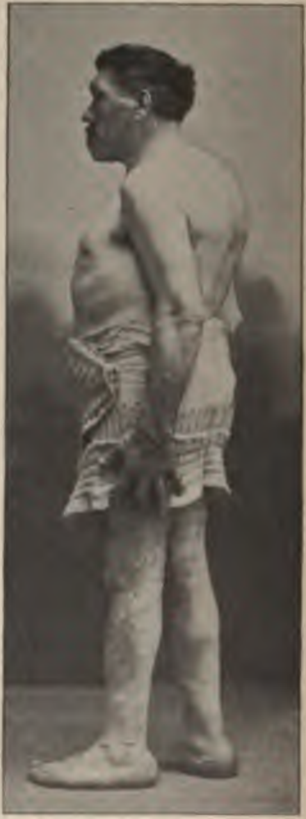
Many cases of giantism are probable cases of acromegaly. There is usually complaint of great weakness. Diabetes has been frequently noted in connection with this affection.

FIG. 65.



Facial hemiatrophy. (LYMAN.)

FIG. 66.



Case of acromegaly. (OSBORNE.)

CHANGES OF ELECTRICAL EXCITABILITY IN MOTOR NERVES AND MUSCLES.

A muscle may be excited to contraction through an electrical stimulus applied to the nerve (indirect excitement) or to the muscle (direct excitement).

If nerve and muscle are healthy the faradic current induces, by either the direct or indirect method, muscular contractions that, with the increase in strength of the current, become tetanic.

For the galvanic current a current reverser is employed, enabling the examiner rapidly and at will, to convert the testing pole into the positive or the negative pole. The pole placed on the sternum or neck is the "indifferent" pole. The negative pole is known as the cathode, the positive as the anode.

If we test a healthy muscle with the galvanic current, gradually increasing in strength, it is found always to react first when there is closure of the cathode—that is, when the current is closed and the testing pole becomes the cathode or negative pole. The continuous and uninterrupted passage of the current does not produce contraction. There is no anodal contraction nor any cathodal opening contraction with a current just strong enough to produce a cathodal closing contraction. If, however, the strength of the current is increased a point is found where contractions are induced by the positive pole, by anodal opening and anodal closing, the current producing the latter being usually a trifle stronger than that required to produce an anodal opening contraction. With the current producing the anodal contractions the cathodal closing contraction is much stronger. As the current is still further increased in strength—and usually producing great pain—the cathodal closing contraction becomes tetanic, the anodal contractions are much stronger, and a contraction also appears with the negative pole over the muscle, as the current is opened—a cathodal opening contraction. The following formula represents the order of appearance of contractions in a healthy muscle, it being understood that the current is gradually increased in strength, and that as each succeeding contraction appears, the ones that have already appeared become stronger and stronger:

Let A = Anode or positive pole; C = Cathode or negative pole; O = Opening; C′ = Closing.

(1) Negative Closing = CC′.
(2) Positive Opening = AO.
(3) Positive Closing = AC′.
(4) Negative Opening = CO.

To represent more fully what occurs we may tabulate as follows, letting type as indicated by the underscoring indicate strength of contraction:

(1) With very weak current—No contraction.
(2) With a moderate current—CC′.
(3) With a strong current—$\underline{CC'}$-AO.
(4) With very strong current—$\underline{\underline{CC'}}$-$\underline{AO}$-AC′.
(5) With strongest current—$\underline{\underline{\underline{CC'}}}$-$\underline{\underline{AO}}$-$\underline{AC'}$-CO.

Electric stimulation of nerves and muscles is an aid not alone to therapy, but to diagnosis and prognosis. Quantitative and qualitative changes are to be noted.

To the faradic current a normal nerve or muscle responds whether negative or positive pole be applied. Increased excitability is seen in tetany, and very early in some cases of peripheral paralysis. The excitability is usually lost or diminished in bulbar paralysis, acute anterior poliomyelitis, progressive muscular atrophy, peripheral neuritis, etc.

With the galvanic current there are not alone quantitative changes, but qualitative as well.

By *reaction of degeneration* is meant a certain qualitative and quantitative change in the electrical reaction of the muscles and nerves in which certain primary or secondary degenerative anatomical changes have occurred.

In a nerve in which degeneration has occurred there is no response to faradic or galvanic current. In the muscle, however, while the faradic excitability is diminished or lost, there is increased excitability to the galvanic current, but with certain changes that constitute the reaction of degeneration. A muscle showing normal faradic excitability will not exhibit reaction of degeneration. Summarized, the reaction of degeneration is characterized by:

(1) Muscular contraction with relatively weak currents.

(2) The muscular contractions are slow and sluggish, not rapid as in a normal muscle.

(3) Mechanical irritability is usually increased; a sharp stroke upon the muscle may provoke a contraction.

(4) Anodal contractions are provoked by weaker currents than cathodal closing contractions. The cathodal opening contraction may even be stronger than the cathodal closing. In other words, the cathodal closing contraction, usually the strongest and first to appear, is postponed, and is weaker than some of the other contractions.

(5) In protracted and in incurable cases even the reaction of degeneration may finally disappear and the muscle remain permanently paralyzed and inactive on strongest electrical stimulation. In cases with more favorable prognosis there is

noted a gradual return of strength to the cathodal closing contraction, the faradic excitability again becomes manifested and a final return to the normal electrical excitability occurs.

Clinical Significance of the **Reaction** of **Degeneration**. Only in those cases in which a muscle is cut off from its communication with the ganglion cells of the anterior gray horns of the cord do we find the reaction of degeneration. It is met with, therefore, in lesions involving these cornua, as in acute anterior poliomyelitis, progressive spinal muscular atrophy, transverse myelitis. Destruction of the spinal nerve, as in neuritis, diphtheritic paralysis, toxic paralysis, likewise produces it, as the communication with the ganglion cells is thus cut off, though they themselves may be intact. It is extremely interesting to note how in a cervical myelitis there is atrophy and reaction of degeneration in the upper extremity muscles, while in the lower extremity muscles, though they are paralyzed, there is neither atrophy nor reaction of degeneration, as the trophic cells of the anterior horns are uninjured.

In paralysis of cerebral origin, in spastic paralysis, no reacaction of degeneration exists. Its presence indicates lesion of the peripheral nerve or the gray matter of the cord. In some mild peripheral paralyses no such reaction can be made out. Here the damage is slight, the prognosis favorable. The more marked and the more lasting the reaction the worse the prognosis.

THE CRANIAL NERVES.

THE OLFACTORY NERVE.

Lesions of the nerve terminations, as chronic nasal catarrh, lesions in the course of the nerve or in the bulb, as from the effects of fracture, caries, meningitis, neuritis, tumor, or lesions of the olfactory centre, believed to be the uncinate gyrus, may produce disturbances of the sense of smell.

A destructive lesion would produce a complete loss of the sense of smell, **anosmia**. Slight alteration, but not absolute destruction of nerve termination, nerve fibre, bulb, or centre, might cause unnatural subjective sensations of smell, **parosmia**. This is sometimes seen in the hysterical, in cases of migraine, tabes, etc. Pleasant odors may be unpleasant. **Hyperosmia** is increased sensitiveness. It is at times seen in nervous, hysterical women.

To test the sense of smell, irritants, as ammonia, should not be employed. Oil of peppermint or some other essential oil should be employed. The nostrils, each one, should be examined carefully, as most cases of anosmia are due to disease of the nasal cavities.

LESIONS OF THE OPTIC NERVE AND TRACT.

In all cases of nervous disease it is important to examine the eye, the movements of the ball, of the lid, of the iris, and the ophthalmoscope is a necessary instrument to the trained neurologist. But a brief outline can here be given of the more important aids to diagnosis to be obtained from an examination of the eye. Fuller details must be sought in the text-books on ophthalmology and on neurology.

Lesions of the Retina.

Retinitis is met with at times in Bright's disease, syphilis, leukæmia, anæmia, occasionally in diabetes, purpura, chronic lead poisoning. Albuminuric retinitis is oftenest found in interstitial nephritis. The retina may show white spots, streaks, or patches, the disk being nearly normal, the degenerative form; or there may be many hemorrhages with few signs of inflammation, the hemorrhagic form; or the retina may be much swollen, clouded, with the disk obscure, the inflammatory form. In anæmia and in malaria with consequent anæmia there may be a neuro-retinitis associated, particularly in pernicious anæmia, with hemorrhages. In leukæmia the retina may show hemorrhages or prominent whitish or yellowish areas.

There are certain functional disturbances of the retina in which no gross changes can be met with. In some cases of uræmia, in poisoning by lead or by quinine, in hysteria, in tobacco amblyopia, these functional disturbances are met with. In the latter affection the loss of sight is usually gradual, and there is a central dark spot, scotoma, for red and green. Atrophy of the disk not infrequently follows. Nyctalopia is a condition in which objects are seen only during the day or by strong light; hemeralopia, a condition in which objects are seen in the shade or in the twilight.

Lesions of the Optic Nerve.

Optic neuritis or choked disk is of great value as an aid in diagnosis, as it is commonly seen in Bright's disease, and is one of the cardinal symptoms of brain tumor. It may also be found in abscess and in meningitis. In the early stages the disk is congested, the edges blurred. Later the congestion is more marked, striation more plainly visible, the physiological cupping disappears, the edges of the disk can no longer be made out; the veins are dilated; hemorrhages may be present. Following optic neuritis there may be atrophy in which the disk has a blank, white appearance, with irregular outlines and small arteries. Optic nerve atrophy is occasionally a primary affection, hereditary, or associated with spinal diseases, particularly tabes. With optic nerve atrophy there is not only dimness of vision, but the field of vision and the color-sense are also altered.

Affections of the Optic Chiasm and Tract.

In attempting to understand lesions as they affect the optic chiasm and tract, one must remember that at the optic chiasm there is a partial decussation of the fibres, so that those from the right hemisphere go to the right half of the retina of either eye; those from the left to the left half of the retina of either eye. This is seen by studying the accompanying diagram. The effects of lesions in different situations would be as follows:

FIG. 67.

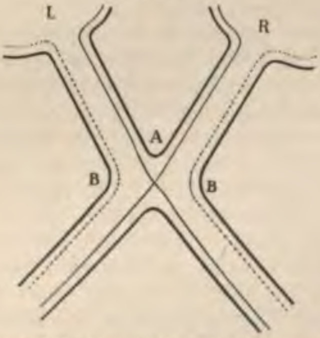


Diagram of the course of the fibres in the optic chiasm. (After STRÜMPELL.)

"A lesion of the optic nerve will produce total blindness of the corresponding eye. A lesion of the optic chiasm, as at A, temporal hemianopia, that is, paralysis of the nasal half of the retina with corresponding loss of the temporal portion of the visual field. If each lateral region of the chiasm is involved, an extremely rare occurrence, as at B and B′, nasal hemianopia will be produced. A lesion of the optic tract between the chiasm and the geniculate bodies, or between the geniculate bodies and the cerebral cortex, or a lesion of the cuneus, will produce a lateral hemianopia. A lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as *mind blindness.*

"Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wernicke's **hemiopic pupillary inaction.** The pupil reflex depends on the integrity of the retina or receiving membrane, on the fibres of the optic nerve and tract which transmit the impulse, and the nerve centre in the geniculate bodies which receives the impression and transmits it to the third nerve along which the motor impulses pass to the iris. If a bright light is thrown into the eye and the pupil reacts, the integrity of this reflex arc is demonstrated. It is possible in cases of lateral hemianopia so to throw the light into the eye that it falls upon the blind half of the retina. If, when this is done the pupil contracts, the indication is that the reflex arc above referred to is perfect, by which we mean that the optic nerve fibres from the retinal expansion to the centre, the centre itself, and the third nerve are uninvolved. In such a case the conclusion would be justified that the cause of the hemianopia was central; that is, situated behind the geniculate bodies, either in the fibres of the optic radiation or in the visual cortical centres. If, on the other hand, when the light is carefully thrown on the hemiopic half of the retina, the pupil remains inactive, the conclusion is justifiable that there is interruption in the path between the retina and the geniculate bodies, and that the hemianopia is not central, but dependent upon a lesion situated in the tract. This test of Wernicke's is sometimes difficult to obtain. It is best performed as follows: The patient being in a dark or nearly dark room with the lamp or gaslight behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus, upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focused from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light." (Seguin.)

Motor Nerves of the Eyeball.

The third nerve supplies the levator palpebræ superioris, the superior, the internal and inferior recti, the inferior oblique, the ciliary muscle, and the constrictor of the iris. An affection of the nucleus is usually associated with disease of the nuclei of other ocular nerves. The nerve itself is usually involved as the result of meningitis, tumors, or aneurism. Where there is paralysis the eye can be moved outward, downward, and inward. There is divergent strabismus, diplopia, ptosis. The pupil does not contract to light; the power of accommodation is lost. In some cases of paralysis of the oculomotor nerve some of the branches may be spared.

Where the ciliary muscle is paralyzed (cycloplegia), there is loss of accommodation. The iris may be paralyzed in one of three ways: There may be (1) loss of reflex contraction of the sphincter on exposure to light. Each eye should be tested separately with the other covered, and the light should be brought suddenly before the eye and at a distance from the eye. (2) There may be loss of the reflex dilatation, ordinarily produced by stimulation of the cutaneous nerves. This is tested by pinching the skin on the side of the neck, which ordinarily produces dilatation of the pupil. (3) There may be loss of the power of contraction of the iris on accommodation, tested by having the patient look at a distant object and then at a near one, both in the same line of vision. The Argyll-Robertson pupil, met with in tabes, is the pupil in which the light reflex is lost, while the accommodation reflex is still present. Spasm in the distribution of the oculomotor nerve may show as a nystagmus, met with in many brain lesions, and one of the cardinal symptoms of multiple sclerosis. Blepharospasm is the spasm of the orbicularis palpebrarum.

THE FOURTH NERVE.

The **fourth nerve or pathetic nerve** supplies the superior oblique muscle; its paralysis, either central or peripheral, causes defective downward and inward movement with diplopia in looking downward.

THE SIXTH NERVE.

The abducens or sixth nerve supplies the external rectus muscle. Its paralysis causes defective outward movement of the eyeball with convergent strabismus and diplopia on looking to the paralyzed side.

Ophthalmoplegia, *i. e.*, a paralysis of all the ocular muscles, may be acute and due to hemorrhage in the region of the nuclei, or chronic, as in tabes and general paralysis. In the latter condition there is paralysis of the levator and superior recti muscles first, and then of the other external ocular muscles. This produces ptosis and immobility of the eyeball, external ophthalmoplegia. Or the pupil may fail to respond either to light or in accommodation, internal ophthalmoplegia. Commonly there is a combination of these two forms, complete or total ophthalmoplegia.

THE FIFTH NERVE.

Anæsthesia of the Trigeminus. The trigeminal nerve may be anæsthetic from a cause which may compress the trunk of the nerve, the Gasserian ganglion or any of its branches. The disease is recognized by an examination of the face and scalp of the patient, and where only one branch of the nerve is affected the corresponding portion of the skin is found anæsthetic. The conjunctiva and cornea being anæsthetic, foreign bodies readily lodge in the eye, are not felt by the patient, excite inflammation, and an ophthalmia is a frequent result. This condition is regarded, however, by some as due to a disturbance of the trophic centre. In trigeminal anæthesia there is usually alteration or diminution of the taste on the anterior two-thirds of the tongue on the affected side. This is the territory supplied by the lingual nerve. This form of anæsthesia is usually unilateral. The skin may be slightly cyanotic, cool to the touch, and the countenance seem somewhat bloated.

Paralysis may be due to a lesion in the pons, to tumor of the base, or to basilar meningitis, or to pressure from tumor, or to destruction of the nerve by inflammation anywhere in its course.

In **neuralgia** of the fifth nerve the ophthalmic branch is most commonly affected, the nerve as it emerges through the supra-orbital notch being sensitive to pressure. Sensitiveness over the infra-orbital foramen, with pain distributed over the cheek, indicates involvement of the second branch. Neuralgia of the third branch is manifested by pain in the lower jaw, the temple, the parietal region, and the tongue. A tender point is found at the inferior dental foramen.

Watering of the eyes and reflex muscular spasms may accompany the sudden, sharp, darting and boring pains of trifacial neuralgia.

Where there is **paralysis** of the *motor* fibres of the fifth nerve the movements of mastication on the affected side are impossible.

Clonic spasms of the muscles supplied by the fifth nerve occur during convulsions, and are seen in the chattering of the teeth during a chill. Clonic spasm, *trismus*, is met with in tetanus.

THE SEVENTH NERVE.

Paralysis may be peripheral, nuclear, cortical.

Peripheral paralysis, Bell's palsy, occurring often after exposure to cold, causes inability to move the affected side of the face, including the frontal muscle. The wrinkles are absent even on attempted movement, as in puckering of the lips, laughing, during which movements the face seems, by contrast, drawn too much toward the sound side. The lower lid droops, the eye is open.

The sense of taste in the anterior part of the tongue will be lost if the lesion is between the geniculate ganglion and the origin of the chorda tympani. In severe cases there is reaction of degeneration.

Nuclear facial paralysis cannot be differentiated from the peripheral form.

Where the lesion is cortical there is usually accompanying complete or partial hemiplegia, there is no reaction of degeneration, the orbicularis palpebrarum and frontalis are spared. It is worthy of note that while voluntary movement of the facial muscles may be impossible, the same muscles may move under the influence of emotion, as in laughing or crying.

Double facial paralysis may be caused by tubercular or osteomyelitic processes in both temporal bones, by a lesion of the pons or base.

Spasm of the muscles supplied by the facial nerve may affect those about the eye, when it is known as blepharospasm, or all the muscles of one or both sides.

THE EIGHTH NERVE.

Paralysis of the auditory nerve may be due to cold ("rheumatic"), to pressure of a tumor or exudate, or to disease of the temporal bone. Disease of the labyrinth is accompanied by vertigo.

Hearing is tested by noting the distance at which a watch or a tuning-fork can be heard, comparison being made with the sound ear or the ear of a person with normal hearing. "If the cause of deafness is middle-ear disease, impacted cerumen, or obstruction of the Eustachian tube, a vibrating tuning-fork placed upon the vertex will be heard much more intensely on the deaf side."—Rinne's test. (Musser.) Where the deafness is neurotic this "bone-conduction" test fails to show increased acuity of hearing on the deaf side.

Hyperæsthesia of the auditory nerve is seen at times in hemicrania, meningitis, hysteria.

Ménière's Disease. *Vide* p. 411.

THE NINTH NERVE.

Loss of taste due to peripheral lesion, as in the coated tongue of dyspepsia and of fevers, is not uncommon. Central lesions of the glosso-pharyngeal nerve are extremely rare.

The sense of taste over the area of distribution of this nerve—the posterior third of the tongue—is tested by placing on the protruded tongue a small amount of a substance that is sweet, sour, bitter, salty, alkaline, etc., and noticing whether the patient, with the eyes closed, can discriminate between the different articles employed.

TENTH NERVE—THE PNEUMOGASTRIC.

Affections of this nerve may be manifested in various ways and by disturbances in organs widely separated, *e. g.*, the pharynx, larynx, lungs, heart, œsophagus, stomach, as the nerve sends branches to all these important organs. It may be affected by nuclear degeneration, by pressure of tumors, exudates, enlarged glands, aneurisms; by neuritis.

Pharyngeal Branches.

If the pharyngeal branches are paralyzed there is difficulty in swallowing, food may pass into the larynx, liquids may be regurgitated through the nares instead of entering the œsophagus.

Laryngeal Branches.

Gowers's table shows the result of the various forms of laryngeal paralysis. It is to be remembered that the motor branches to the larynx come from the internal branch of the spinal accessory nerve:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive, little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Laryngeal Spasm.

Laryngeal spasm is met with in the laryngeal crisis of tabes and in the spasmodic croup of children. It is a spasm of the adductors. The child usually awakes from sound sleep struggling for breath, cyanotic, hoarse. A deep, crowing inspiration marks the end of the spasm.

Cardiac Branches.

The sensory affections of the heart, such as palpitation, pain as in angina pectoris, are probably conveyed by this nerve to the brain. Irritative lesions of the nerve produce a slowing of the heart's action. The bradycardia following acute infectious diseases is in most instances a neurosis. Paralysis of the nerve, as from pressure of an aneurism, tumor, or from nuclear disease, or from neuritis as in post-diphtheritic paralysis, causes the removal of the inhibitory influence of the nerve, resulting in tachycardia.

Pulmonary Branches.

But little is definitely known concerning lesions of these branches. Asthma is believed, in some cases at least, to be a neurosis of the bronchial branches.

Œsophageal and Gastric Branches.

Spasm of the œsophagus, or paralysis, indicates irritative or destructive lesions of these branches. Gastralgic attacks are probably neuralgias of the terminal gastric fibres. Vomiting may be the result of direct stimulation of the nerve, as in meningitis, or may be reflex.

THE ELEVENTH NERVE.

If the **spinal accessory** nerve is paralyzed the sterno-mastod of the affected side and a portion of the trapezius are affected. The shoulder droops, the scapula is rotated inward, the arm is raised only with great effort, the head is turned with difficulty toward the sound side.

Spasm of the spinal accessory (torticollis) may be congenital—in which case there is usually atrophy of the face on the corresponding side—or acquired. Clonic spasm is attended by a sudden, often painful, jerking of the head toward the affected side. With tonic spasm the face and chin are permanently turned toward the sound side, the occiput being drawn by the tense sterno-mastoid toward the affected side. The trapezius and other muscles may be affected as well as the sterno-mastoid.

TWELFTH NERVE.

Where paralysis of the hypoglossal nerve is due to disease of the nucleus, as in bulbar paralysis, there is atrophy of the tongue, usually bilateral, reaction of degeneration, inability to move the organ, and consequent difficulty in talking and swallowing. Disease above the nucleus is unilateral, attended by no atrophy or degenerative reaction. The tongue is pushed, when protruded, toward the affected side. Peripheral paralysis would be attended by atrophy and degeneration.

THE SPINAL NERVES.

NEURITIS.

General Symptoms of Neuritis.

The symptoms common to a neuritis are pain, tenderness, swelling, vasomotor disturbances, paræsthesiæ, atrophy, reaction of degeneration, paralysis. These vary, of course, according to the severity of the inflammation, its exciting cause, the duration of the affection.

Pain is usually marked, boring, darting, or burning in character, and increased by movement. Tenderness on pressure, and swelling, can often be made out in accessible nerves. Paræsthetic numbness, tingling, burning, sense of weight are not uncommon. Vasomotor disturbances are seen in œdema, redness, glossy skin, dryness of the nails, local sweating, etc. When the inflammation is of severe grade there is atrophy of the muscle, with consequent paresis or paralysis. The muscle exhibits in varying degrees the degeneration reaction.

By **neuralgia** is meant a pain in the region of distribution of a nerve, without the signs of a nerve inflammation being present. Many neuralgiæ are probably neuritic in character. In other cases local causes, as pressure, neuromata are at fault. General debilitating conditions predispose to neuralgic pains, *e. g.*, anæmia, chlorosis, diabetes. The diagnosis between neuralgia and neuritis is based on the fact that in the former pain is oftener paroxysmal, tenderness is most marked in certain localities (points of tenderness), there is no enlarged, tender nerve trunk to be made out, there is no blunting of sensation, vasomotor disturbances, atrophy and paralysis are not met with.

Neuritis in Special Nerves.

A few only of the commoner and more important nerves affected by inflammation, with an epitome of the symptoms and signs, can be mentioned:

1. **Posterior Thoracic Nerve.** Paralysis of the serratus magnus; vertebral border of scapula lifted from body when arm is moved forward; scapula therefore assumes a wing-like position.
2. **Suprascapular Nerve.** Paralysis and atrophy of supra- and infra-spinatus muscles; external rotation of humerus lost.
3. **Circumflex Nerve.** Deltoid paralysis, inability to raise

arm, loss of rounded contour of shoulder when atrophy is present.

4. **Musculo-cutaneous Nerve.** Paralysis of biceps and brachialis.

5. **Musculo-spiral Nerve.** Paralysis of extensors of wrist and fingers, with wrist-drop and loss of supination. Wrist-drop in lead palsy usually bilateral. Triceps may be affected.

6. **Median Nerve.** Paralysis of flexors of fingers and thumb, the pronators, abductor pollicis. Wrist turned toward ulnar side; flexion of second phalanges on first lost.

7. **Ulnar Nerve.** The flexor carpi ulnaris, ulnar portion of the flexor profundus, the interossei, the adductor pollicis, the muscles of the little finger, are paralyzed, and sensation is also affected.

8. **Sciatic Nerve.** Common and often classed as a pure neuralgia. Pain and tenderness down the back of thigh from the small of the back. In cases pain extends to ankle. Pain is boring or dull. May be paroxysmal. Leg feels heavy and numb. Leg flexed; movement often painful. Atrophy may or may not occur.

This condition is to be differentiated from inflammation of the hip-joint, tubercular or simple inflammatory, from pain in the sciatic nerve due to pelvic disease, by careful physical examination, if need be, under an anæsthetic. The pains of tabes are sometimes misleading and should be kept in mind.

Local and Multiple Neuritis.

Neuritis is either localized or general.

Localized neuritis may arise from cold, traumatism, or the extension of inflammation from neighboring parts, as when there is facial neuritis from inflammatory processes in the temporal bone. The symptoms are almost exclusively local. There is pain along the course of the affected nerve, which is extremely tender to pressure and can sometimes be felt as a hard cord. The skin may be reddened or œdematous. Numbness and tingling are complained of, and the sense of touch is blunted. Trophic disturbances in the shape of sweating, herpes, glossy fingers, and dryness of the nails are seen in cases. The muscles may atrophy. Contractures occur. Occasionally the neuritis in a single nerve seems to extend upward and involve the spinal cord, or through some unknown avenue implicate the corresponding nerve on the opposite side, a "sympathetic" neuritis. In a severe neuritis the reaction of degeneration can be made out.

Multiple neuritis may come on during or following acute infectious diseases, such as diphtheria, typhoid, and the influenza; may be due to arsenical and lead poisoning; may be an endemic infectious disease, beri-beri; may be due to chronic alcoholic poisoning; or may in some instances come on as an acute idiopathic affection following, perhaps, exposure to cold or overexertion.

In all these cases the symptoms are very similar to those described under the head of localized neuritis, save that many nerves are involved, most commonly those of the extremities. The nerves are tender to pressure, there is usually severe pain, loss of muscular power, which results frequently in wrist-drop and foot-drop, and in a flaccid paralysis. The muscles waste. There is reaction of degeneration. The fingers and the feet are swollen and have glossy skin. In some cases the nerves of the eye may be involved, or the pneumogastric nerve. The latter condition is, of course, dangerous to life, disturbing as it does the functions of the larynx, the heart, the stomach, and the œsophagus.

In the acute febrile variety there may be the symptoms of an infection in chills, pain, high temperature, loss of appetite, and vomiting. The alcoholic variety comes oftenest in steady old topers. It is chronic in its onset, chronic in its course. Fever is rare. Convulsions, delirium, and hallucinations closely resembling those of delirium tremens are not infrequent. Following this variety, as also following a diphtheritic neuritis, where it involves the lower extremities, there is the peculiar *steppage* gait resembling somewhat the gait of the ataxic. The foot is drawn forcibly forward with the toe lifted high; the heel is brought down first, then the entire foot. The patient walks as though he were stepping over obstacles. In a multiple neuritis following diphtheria, as is well-known, the nerves of the pharynx, of the eye, are oftenest affected, though many peripheral nerves may be involved. In the neuritis of lead poisoning the nerves most frequently affected are those of the extensors of the wrist, so that wrist-drop is the common form of paralysis.

Beri-beri is an acute infectious disease met with in India, China, and Japan, and presenting several types. Its onset is with fever, anæmia, and general anasarca. In other cases there is great numbness, loss of tendon reflexes, areas of anæsthesia, muscular atrophy, and anasarca. Its occurrence in epidemic form, the marked sensory disturbances, with ensuing paralysis, the œdema, should call attention to the possibility of the disease being beri-beri.

The diagnosis of multiple neuritis is not always easy. It has been mistaken for *rheumatism*, but even a hasty examination should show that the pain is along the course of the nerves and not in the joints.

It can readily be mistaken for *myelitis*, but in myelitis pain is less marked, or is entirely absent. The paralysis usually comes on more suddenly. There is involvement of the bladder and rectum, and these organs are usually spared in peripheral neuritis, and in the cervical and dorsal forms the reflexes are exaggerated. If the myelitis is a lumbar affection the diagnosis is more difficult or even impossible, unless we have a clear history of the case in the earlier stages, showing the pain and tenderness along the course of the nerves, for in both forms of disease there would be paralysis, atrophy, reaction of degeneration, and there might be involvement of the bladder and rectum. This latter fact, however, would speak in favor of myelitis and against peripheral neuritis.

The *steppage* gait and loss of patellar reflexes would make one think of *tabes*, but true inco-ordination is not usually present. Romberg's symptom is absent; foot-drop and other forms of paralysis are uncommon in locomotor ataxia, and in neuritis by the time the *steppage* gait had been attained there would be no lightning pains. The pupillary symptoms would also be absent. The outlook in peripheral neuritis is generally good, though the possibility of resulting contractures, of permanent paralysis, or of the involvement of vital nerves should not be forgotten.

SPINAL LOCALIZATION.

Transverse lesions produce paralysis and anæsthesia in the parts below the segment destroyed (paraplegia). The bladder and rectum are also paralyzed. If the lumbar enlargement is involved there is loss of knee-jerk and no spasticity. If a segment above the lumbar enlargement is involved the patellar reflex is increased, ankle-clonus is present, and the paralysis is spastic. The table of M. Allen Starr and the diagram from Gowers will enable one to locate accurately the lesion in a given case.

FIG. 68.

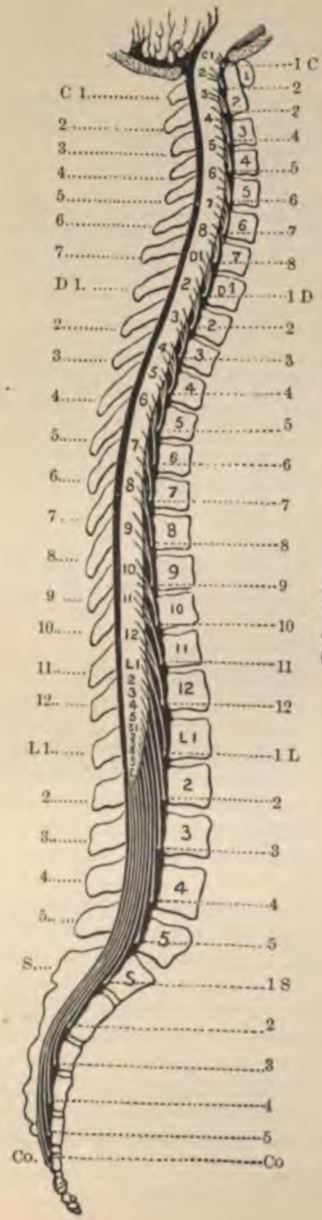


Diagram showing the relations of the bodies and spines of the vertebræ to the segments of the cord and to exits of the nerves.—(GOWERS.)

Segment.	Muscles.	Reflex.	Sensation.
II. and III. c.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. c.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V. c.	Deltoid. Biceps. Coraco-brachialis. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. c.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. c.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. c.	Flexors of wrist and fingers. Intrinsic muscles of hand.	……	Forearm and hand, inner half.
I. D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.	……	Forearm, inner half. Ulnar distribution to hand.
II. to XII. D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary regions causes retraction of epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.

Segment.	Muscles.	Reflex.	Sensation.
I. L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak) Quadriceps femoris.	Patella tendon. Stroking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.	....	Front and inner side of thigh.
IV. L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier) Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.	....	Back of thigh, back of leg, and outer part of foot.
I. to II. S.	Flexors of ankle. Long flexor of toes. Peronæi. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh, leg, and foot, outer side
III. to V. S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion, ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

A unilateral destructive lesion produces paralysis on the same side as the lesion, anæsthesia on the opposite side.

Disease of the **antero-lateral columns**, the motor tract, results in partial or complete paralysis with increased reflexes, spastic condition of muscles. Pain is absent. Sensation remains normal.

When the posterior external columns are diseased, as happens in locomotor ataxia, there are pain, paræsthesiæ, incoordination, and loss of reflexes. The muscles lose nothing in power.

Anterior horn disease results in atrophy, with reaction of degeneration, loss of power, abolition of reflexes.

A lesion of one tract or area of the cord is known as a systematized lesion, as when the lateral columns alone, or the anterior horns alone, are involved. Often, however, there are combined system diseases. Thus the lateral columns with the anterior horns may be affected, as in amyotrophic lateral sclerosis. Here to the symptoms of lateral column disease would be added those of anterior horn disease, atrophy, reaction of degeneration, etc. In other diseases, as in multiple cerebro-spinal sclerosis, the lesion is indiscriminate, involving irregularly various systems or parts of systems and at various levels of the cord.

THE SPINAL MENINGES.

SPINAL MENINGITIS.

The meninges of the cord are very rarely primarily affected except in epidemic cerebro-spinal meningitis. They may, however, be acutely inflamed secondarily to an inflammatory process in the immediate neighborhood, or from the localization of a specific poison of a disease, as, for instance, during the course of some infectious disease, as typhoid, scarlet fever, pyæmia. The pia mater is the membrane chiefly affected, and by spinal meningitis is meant, unless otherwise specified, a leptomeningitis. Tuberculosis may involve the meninges of the cord, as those of the brain.

In attempting to diagnose a spinal meningitis we should always seek for the primary source of the infection. It is very rare to have the spinal meninges affected without also the involvement of the cerebral meninges. Fever, malaise, disturbance of appetite, etc., may be due to the primary affection or to an inflammatory process that is primary in the cord. Pain is a prominent symptom. There is sensitiveness on pressure over the spine; movement of the spinal column is resisted by the patient who holds the back stiff. As the basilar meninges are involved there is usually retraction of the head. Pains shoot from the cord down the course of the spinal nerves into the extremities; there may be hyperæsthesia and other disturbances of sensation, as tingling and formication. Muscular twitchings, muscular rigidity, may be present. The reflexes are oftentimes abolished. There may be disturbances of the bladder and of the rectum. Paresis or paralysis may occur if the exudate is large, and if by pressure it destroys the function of the cord, or if the inflammatory process invades the cord substance itself. The seat of the lesion can be determined by noting the most painful points of the vertebral column and the height to which cutaneous hyperæsthesia and pain extend. If the meninges of the medulla and of the base of the brain are involved the symptoms will be those of a cerebro-spinal meningitis.

PACHYMENINGITIS CERVICALIS HYPERTROPHICA.

This disease is described by Charcot as due to a chronic thickening of the dura in the cervical cord. The symptoms result from pressure upon the nerve roots, of the cord itself in this region, with a secondary descending degeneration of the pyramidal tract of the cord. At first there is severe pain in the arms, and a feeling of numbness is noted. Occasionally there is herpes. Soon paralysis begins, attended by atrophy. The flexors are chiefly affected, so that the hand assumes the shape of a claw-hand. The muscles show the reaction of degeneration. When secondary descending degeneration of the lateral columns occurs the symptoms of spastic paralysis, increased tendon reflexes and rigidity of muscles, appear. Late in the course of the disease there may be complete paraplegia, preceded, perhaps, by paralysis of the bladder and of the rectum. The disease is to be distinguished from amyotrophic lateral sclerosis by the fact that in the latter disease there are no disturbances of sensibility; that the atrophy extends to the lower extremities; and by the fact that there are, sooner or later, bulbar symptoms. In amyotrophic paralysis the bladder is not affected.

THE BLOODVESSELS OF THE CORD.

Anæmia, acute and passive *hyperæmia* of the spinal cord are rather pathological findings than clinical entities. *Embolism* and *thrombosis* are rarely diagnosed and are usually but the consequents or the concomitants of other disease of the cord, as an endarteritis of the syphilitic or of the aged. There is really no disease that can clinically be recognized as a spinal anæmia, congestion, embolism, or thrombosis.

HEMORRHAGE INTO THE CORD, OR HÆMATOMYELIA.

"Primary hemorrhage into the spinal cord is as rare as hemorrhage into the brain is frequent." (Strümpell.) Traumatism, cold, overexertion occasionally produce hemorrhage into the cord. Hemorrhages may occur in connection with myelitis, and in some cases it is questionable which is primary, the hemorrhage or the inflammatory trouble.

When there is a spinal apoplexy the onset of symptoms is sudden; there is pain in the back; sudden loss of power in the body below the seat of the hemorrhage. Sensation is lost; the reflexes disappear. Later the clinical picture is identical with that of myelitis, with paraplegia, loss of reflexes if the lumbar cord is involved, exaggerated reflexes if the dorsal or cervical cord is implicated. The bladder and réctum may be affected as in myelitis. Recovery, from absorption, sometimes occurs.

CAISSON DISEASE.

Those who work in a condensed atmosphere, on going suddenly to a rarer one, are sometimes affected by a peculiar disease that is probably due to a hemorrhage into the cord, chiefly in the lumbar region. There is usually a sudden pain, most marked in the legs and abdomen. The patient becomes dizzy, vomits, may become comatose. Quite suddenly or gradually there is loss of power in the legs, and in some cases in the arms also. Death may result, the patient dying comatose. In most cases, however, the outlook is fairly good, and recovery is complete, or there may be some permanent paralysis and atrophy. The diagnosis presents no difficulty when we have a clear history of the sudden onset of the disease, with pain, loss of motion, following the change from a dense to a rarer atmosphere.

THE SPINAL CORD.

I. ACUTE AFFECTIONS.

TRANSVERSE MYELITIS.

In transverse myelitis the gray and white matter of the cord at some level are inflamed; occasionaly the gray matter seems to be solely or chiefly involved, and a central myelitis is spoken of; at times, also, several segments of the cord are involved—a diffuse myelitis.

The causes of myelitis are: 1. Cold and overexertion. 2. Trauma, as, for instance, fracture, dislocation. 3. Infectious diseases, as smallpox, diphtheria, measles. 4. Pressure, as from tumor or gumma, though here the inflammatory process in the cord is not well marked, the process being rather a degeneration than an inflammation.

The acute form of the disease is characterized mainly by evidences of **motor disturbance**; the paresis of the lower extremities may be quite suddenly developed and rapidly lead to complete paraplegia. If the cervical segment is involved the upper extremities will become paralyzed as well as the lower. In some cases of cervical myelitis the lower extremities still retain a certain amount of motion. Twitching of the limbs and spasticity are sometimes found.

Sensory disturbances are usually insignificant as compared to the motor disturbance. There may be some pain; often there is complaint of formication, tingling, numbness. A girdle sensation may be noted. Areas of partial anæsthesia may be found, or the entire paralyzed portion may be anæsthetic. At the level of the lesion there is a zone of hyperæsthesia. As a rule, it may be said that the greater the pain, the greater is the involvement of the posterior horns and the posterior columns. The limit of sensation may be of great value in determining the height of the lesion in the cord.

Trophic disturbances are rarely absent. From disuse the muscles atrophy, and if the anterior horns are affected there will be atrophy and the reaction of degeneration. Bedsores readily develop; urticaria and hyperidrosis are frequently met with.

The **reflexes** in transverse myelitis are increased. If, however, the lumbar segment is involved in the inflammatory process they will be lost, as the reflex arc is thus destroyed. The **bladder** and **rectum** become paralyzed, there being at first retention of urine and feces, and later incontinence. This is true, whether the lumbar portion of the cord be involved or not, though very early vesical and rectal symptoms speak for a lumbar involvement. Death in transverse myelitis may occur from an extension of the inflammatory process upward; from cystitis, bedsores, or from intercurrent diseases. Where the cervical region is involved and the upper extremities are paralyzed, there may be, in addition to the other manifestations, vomiting, hiccough, bradycardia, myosis, dysphagia, dyspnœa, syncope.

Transverse myelitis is to be distinguished from Landry's paralysis by the marked sensory disturbance, the trophic changes, the involvement of the rectum and bladder. From acute neuritis it is distinguished by the complete anæsthesia, which is so often present; by the more rapid atrophy, by the fact that the bladder and rectum are involved, and the frequent occurrence of trophic phenomena, as, for instance, bedsores.

LANDRY'S PARALYSIS.

Landry's paralysis is probably an acute infectious disease, possibly a rapidly ascending polyneuritis extending to the spinal cord. The disease begins with general malaise and fever; diarrhœa may be present. Within a few days there is noted a weakness of one leg, or of both; the patient takes to bed, and the paresis, rapidly becoming a paralysis, extends upward, involving the trunk and the arms. Bulbar symptoms, such as difficulty in swallowing and in speaking soon occur. Death follows as soon as there is paralysis of the muscles of respiration. This usually occurs within ten days. Rarely the disease reaches a standstill, and there is a gradual partial or complete recovery. The paralyzed muscles are flabby; the reflexes are diminished or lost; pain is seldom extreme. There may be partial anæsthesia. There is no disturbance of the bladder or rectum. Profuse sweatings may occur. Acute splenic tumor and slight albuminuria have frequently been noted as in other acute infectious diseases.

The rapid progress of the paralysis from below upward, the lack of sensory symptoms, the loss of reflexes, the intact bladder and rectum, with the fever and constitutional evidences of an acute infectious disease make the diagnosis clear.

ACUTE ANTERIOR POLIOMYELITIS.

The gray matter of the cord is rarely affected, except in the anterior horns. The commonest disease of the gray matter is the acute inflammation of the anterior horns, resulting in a later atrophy of the horn and in destruction or degeneration of the large motor cells, with consequent atrophy of nerve roots and wasting of muscles. From the location and character of the lesion, the affection is known as **acute anterior poliomyelitis**; from its attacking almost exclusively young children, **infantile spinal paralysis**. It is rare after the age of five.

The paralysis is strikingly sudden in its onset, and involves usually one extremity, as a leg. Both lower extremities may be paralyzed, or a leg and an arm, even all four extremities. The bladder and rectum remain undisturbed. The muscles of the face are not attacked. Preceding the paralysis there are for a few hours, or perhaps two or three days, slight prodromata or, as in most cases, marked evidence of a systemic infection as shown by fever, restlessness, vomiting, even convulsions. The paralysis remains stationary or shows gradual improvement. After weeks or years certain groups of muscles may resume their functions in a perfectly normal manner. Others remain flabby and atrophied. From the action of normal muscle groups where the antagonists are paralyzed, deformities, as talipes, frequently result. The leg may also be shorter than its fellow, as bony growth is often retarded.

Pain is absent in infantile paralysis, sensibility is perfect. The skin of the affected member is cold and bluish. The reflexes are lost. Within one or two weeks the reaction of degeneration is present in the muscles that are to remain permanently paralyzed and atrophied.

The differential diagnosis rarely presents difficulties save in the early stages. Here the fever, vomiting, convulsions, may lead to the suspicion of a gastric affection or to any of the numerous causes of infantile eclampsia. An examination for paralysis will, after a few days at least, clear up the diagnosis.

Later, the stunted limb with cool, cyanotic skin and with flabby and powerless muscles, the loss of reflexes, the reaction of degeneration, the absence of mental, vesical, or rectal symptoms, the intact sensation, make a picture quite readily recognized.

From **multiple neuritis** it is distinguished by the fact that in multiple neuritis there is (1) slower onset of paralysis; (2) longer duration and greater variability of fever; (3) greater pain, and particularly on movement of, and pressure on, the affected limb; (4) more disturbance of sensation, *e. g.*, numbness, feeling of weight, etc.; (5) more or less swelling and œdema; (6) often involvement of the cranial nerves; (7) later usually more complete recovery.

Cerebral palsy is usually one-sided, may involve cranial nerves, is attended and followed by mental symptoms, *e. g.*, aphasia, idiocy, epilepsy. The reflexes are usually not abolished but exaggerated; there is no reaction of degeneration, post-paralytic athetoid and choreic movements are common; contractures may occur.

Acute anterior poliomyelitis in adults is a rather rare affection. Yet it is met with occasionally after the acute infectious diseases, particularly measles; also following traumatism and exposure to cold.

The phenomena are those of the infantile form, initial chill, fever, rapidly developing paralysis, loss of reflexes, reaction of degeneration, atrophy. The fever may continue longer before the paralysis is manifested, than in the juvenile form, and the muscles involved may include more groups than are commonly affected in the infantile disease. So, too, the improvement is apt to be slower. Pain in the back is sometimes quite severe. The disease, then, is practically identical in its symptomatology with infantile paralysis, save that it is less rapid in its onset and progress. It is most liable to be mistaken for the rapidly ascending paralysis of Landry, for multiple neuritis, or hæmatomyelia. In the latter accident, however, there is absence of fever, the onset is more abrupt, the sphincters rarely escape.

Some authors describe a **chronic anterior poliomyelitis**, which is to be differentiated from the ordinary progressive muscular atrophy on the one hand and from multiple neuritis on the other.

The disease is characterized by a rather slowly progressive and painless paralysis of groups of muscles, followed by atrophy, electrical changes, loss of reflexes. Some cases go on to a fatal issue through bulbar paralysis, others remain at a standstill, while others, after months or years, show improvement.

Lack of known causes of neuritis, absence of pain, swelling, tenderness, distinguish the malady from polyneuritis. The involvement of groups of muscles rather than single muscles, the commencement of the disease in other muscles than those of the hand, the fact that paralysis precedes the atrophy, enable one to differentiate it from progressive muscular atrophy.

The more these cases are studied, however, the more are authors inclined to regard them as cases of multiple neuritis or of progressive muscular atrophy, and some even deny the existence of any separate spinal disease that should go by the name of chronic anterior poliomyelitis, except the progressive atrophy.

II. CHRONIC AFFECTIONS.

Primary Degenerations of the Motor Tract.

The motor tract may be the seat of chronic sclerotic changes, either in the gray matter of the anterior horns, in the lateral columns, in the medulla, or in the cortical substance.

The diseases thus produced vary in their manifestations according to the location of the lesion, and yet are closely related pathologically and not infrequently associated clinically. Where the lesion is solely in the anterior horns, progressive muscular atrophy results; in the lateral colums, lateral sclerosis or spastic paralysis; in the medulla, bulbar or glosso-labial-laryngeal paralysis. A common combination is that where the lateral columns and the anterior gray matter are involved, producing the amyotrophic lateral sclerosis of Charcot. Probably in all cases of lateral sclerosis the anterior horns are somewhat involved.

LATERAL SCLEROSIS.

Most, if not all, cases of lateral sclerosis, *i. e.*, degeneration of the pyramidal tracts, are combined with similar changes in the anterior horns. To the mixed form of disease the name amyotrophic lateral sclerosis was given by Charcot. Clinically all varieties and combinations are met with of atrophy due to the anterior horn lesion, and spasticity due to the lateral column lesion.

In pure **lateral sclerosis (spastic spinal paralysis)** the lower extremities become gradually weak, particularly on exertion. A feeling of stiffness, as if the tendons were too short, is felt. Some rigidity is noticed on passive motion, and the skin and tendon reflexes are exaggerated. While the muscles retain their normal volume, there is such a weakness through rigidity, that a fully developed spastic paresis is developed. The gait is slow, the feet, particularly the toes, seem loth to leave the ground. The patient seems to shove himself forward by short, jerky steps. The upper extremities may later be involved. Sensibility is unaltered, pain is generally slight. The functions of the bladder and rectum are seldom interfered with. The disease may last for ten to twenty years.

Many of these cases of lateral sclerosis are but the beginnings of some other affection as multiple sclerosis, chronic myelitis, ataxic paraplegia, amyotrophic lateral sclerosis. To quote Oppenheim: "One must ever be on the alert against this deception; must ever strive to see whether one may not unmask the so-called spastic spinal paralysis. Every sign that does not properly belong to the symptom-complex of spastic paresis is an indication that another disease is present. And in particular does multiple sclerosis in its early stage, which may last for several years, delight to clothe itself in the garb of the spastic spinal paralysis."

PROGRESSIVE MUSCULAR ATROPHY.

There are no etiological factors discoverable in most cases of progressive muscular atrophy. The disease begins insidiously. Usually the patient notices a weakness in one or both arms, but suffers no pain or other disturbances of sensation. The atrophy generally begins in the thenar and hypothenar eminences. The ball of the thumb flattens out and the thumb is adducted. The interossei and lumbricales atrophy so that the spaces on the back of the hand are sunken, the palm is flattened and the hand has a claw-like appearance. From the hand the atrophy may extend to the forearm or may skip these muscles and invade the deltoid. Months or years may be occupied in these changes. The trunk muscles are rarely affected until late, and those of the lower extremities very late or not at all. If the respiratory muscles, including the diaphragm, become involved, or if the medulla is invaded, giving rise to bulbar paralysis, death follows. Otherwise the patient may live for years, almost literally a living skeleton.

The diagnosis is made on the following points: (1) Atrophy begins in the muscles of the hand; (2) fibrillary twitchings are present in the muscles undergoing atrophy; (3) there is no pain and no altered sensibility—touch, pressure, temperature; (4) electrical excitability to galvanic and faradic currents gradually decreases and may be lost. Reaction of degeneration is only met with here and there; (5) tendon reflexes for the affected area are lost; (6) genuine trophic disturbances of the skin are rare, though from disuse and consequent poor circulation the hands are usually cold and bluish; (7) vesical and rectal symptoms are not present.

AMYOTROPHIC LATERAL SCLEROSIS.

Fright, cold, overexertion, trauma, have been assigned as causes of amyotrophic lateral sclerosis. It is oftenest met with in middle age. The symptoms are, in reality, those of a combination of progressive muscular atrophy, lateral sclerosis, and often in the terminal stage, at least, bulbar paralysis. In some cases the progressive invasion of the various portions of the motor tract can be followed, as in Osler's case, where the disease evidently began in the gray matter, extended to the pyramidal tracts, then to the bulbar nuclei, and later, apparently involved the motor cells of the cortex.

If beginning in the upper extremities, weakness and atrophy of the arm are noted. Fibrillary tremors are common, pain is usually insignificant. If commencing in a lower extremity, rigidity of the muscles is a prominent feature, as well as weakness. In the upper extremity, rigidity, while present, is rarely so well marked as in the lower; the atrophy is atonic. Atrophy is seldom as well marked in the lower as in the upper extremities, and when present is usually tonic. The cutaneous and tendon reflexes are exaggerated. The sphincters are rarely affected. The gait is the slow, stiff-legged, quickstep gait of lateral sclerosis. Bulbar symptoms may appear later, with the characteristic changes in articulation, difficulty in swallowing, atrophy of muscles of the tongue, lips, etc., described under the head of bulbar paralysis. The diagnosis is made upon the combination of atrophy, rigidity, paresis, absence of sensory symptoms, exaggeration of reflexes.

PROGRESSIVE BULBAR PARALYSIS.

Progressive degeneration and atrophy of the nuclei in the medulla oblongata give rise to a well-recognized disease, known as bulbar, or *glosso-labio laryngeal paralysis.* As the latter name indicates, there is paralysis—and usually in the order indicated—of the muscles of the tongue, the lips, the larynx and the pharynx. Pain is absent. Attention is first directed to the difficulty in pronouncing certain letters, such as *l*, *s*, *d*, *t*, for the proper pronunciation of which the tongue has to be employed. This, through absence of aphasic symptoms, is seen to be entirely traceable to faulty innervation of the lingual muscles. Fine fibrillary tremors are seen in the tongue. The lingual muscles atrophy, the tongue grows thin and flabby, wrinkled or furrowed, and its movements, as in articulation, mastication, swallowing, more and more interfered with.

By this time the muscles of the lips have begun to waste, often with a subjective sensation of rigidity or tension. Their movements are restricted. Efforts to pucker the lips, as in whistling or in the pronunciation of certain letters, as *o*, *p*, *f*, *b*, are followed by poor results. The skin over the atrophied and thin lips is wrinkled and furrowed.

The muscles of the pharynx and larynx are next invaded. The soft palate becomes inactive, permitting of regurgitation of food. A nasal twang to the voice is perceptible. The act of swallowing, through weakness of the lingual muscles, is rendered still more difficult through paralysis of the pharyngeal constrictors.

The laryngeal paralysis can be detected with the laryngoscope. But it may be inferred from the altered voice. The production of high notes and of modulations of tone are impossible. The voice is weak and monotonous, and later hoarse and whispering. Food is liable to be swallowed the wrong way.

This complexus of symptoms with the regular, slow progession from tongue, to lips, to pharynx and larynx, renders the diagnosis usually easy. Among other facts that may aid in diagnosis are the following: Late in the disease, reaction of degeneration can sometimes by careful examination be made out. The faucial reflex is usually diminished. Rarely an increase in the jaw reflex can be demonstrated by tapping on the lower jaw with the patient's mouth partly opened. Sensation is generally unimpaired. Salivation is pronounced, both because the patient has lost the power of completely closing the mouth, so that the saliva drips almost constantly, and because of an increased secretion. These patients show no deterioration of mental faculties.

PRIMARY MUSCULAR ATROPHIES.

While not properly belonging under the head of diseases of the cord, primary muscular atrophies are, for convenience, here described with those of spinal origin.

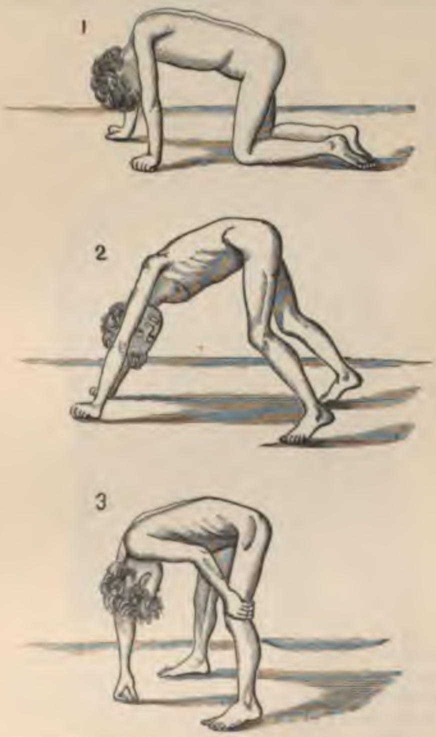
There are two types of primary muscular atrophy: (*a*) the one where there is a pseudo-hypertrophy due to the hypertrophy of some muscular fibres and to the development of fatty and connective tissue between the muscular fibres; and (*b*) the true atrophy, with diminution in the size of separate fibres, but with a minimum amount of adipose and connective tissue. By primary muscular atrophy is meant the muscular atrophy that does not depend upon a lesion of the brain, of the cord, or of the nerves—an atrophy, in other words, that depends upon some primary trouble in the muscle itself.

Various types of muscular atrophy have been described, the chief of these are the following:

(1) **Pseudo-hypertrophic muscular paralysis.** This is a disease oftenest affecting males, frequently showing a history of heredity, and beginning almost invariably before the age of ten years. The child is noticed at first to have some difficulty in walking or in getting up, or to walk with a peculiar gait.

The lower extremities and the loins are found upon careful examination to be weak; the muscles of the back are shrunken, and the child, in order to balance himself, stands with the shoulders thrown backward and the abdomen prominently forward, so that there is marked lordosis. The pseudo-hypertropy is often particularly well marked in the muscles of the calf of the leg, and the contrast between the voluminous muscles and the very evident weakness of the calf of the leg is striking. This hypertrophy, it must be remembered, is but a false hypertrophy. The increased volume is due largely

FIG. 69.

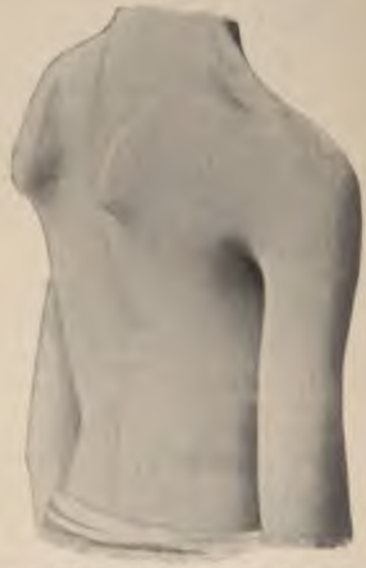


Showing manner in which child with pseudo-hypertrophic paralysis gets up from floor. (GOWERS.)

to the over-development of adipose and connective tissue. Some of the muscular fibres may be hypertrophied, but many of the others are atrophied, so that the total effect is a marked weakness of the muscles.

The peculiar manner in which one of these little patients after lying down, gets up, is almost of itself pathognomonic. It is excellently illustrated by the figures of Gowers. The patient rolls over on the abdomen, gets on all fours, and then, by placing one hand and then the other upon the knee, climbs up his own legs, and gradually, with great effort, attains the erect position.

Fig. 70.

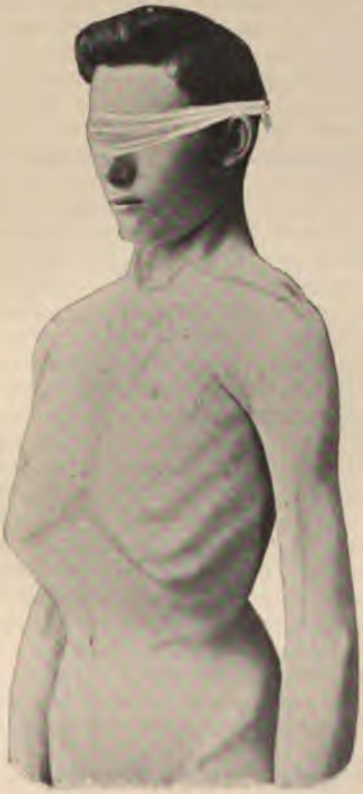


Erb's juvenile muscular atrophy. Shows atrophy of trapezii and wing-like projection of scapulæ, owing to atrophy of serrati. Male, aged twenty-one; disease began at fourteen. (From photograph kindly furnished by Drs. E. S. Pettyjohn and S. R. Slaymaker.)

These patients suffer no pain; there is no disturbance of the rectum or bladder; the reaction of degeneration cannot be made out. Gradually other muscles than those of the lower extremities are involved, and death may occur, after the patient has become bedridden, from involvement of the diaphragm or from intercurrent disease.

(2) **Erb's juvenile** or **hereditary atrophy** begins before the age of twenty, and there is very often a history of heredity,

FIG. 71.



Same case as Fig. 59. Shows wasting of pectorales, drawing up of upper angles of scapula by the levator anguli scapulæ and upper portion of the trapezius, owing to absence of opposition. Also shows wasting of deltoid and biceps. Patient has taken deep inspiration and is holding his breath.

or of the disease in other members of the family. The muscles first showing atrophy are those of the shoulders and of the upper extremities. The order is given as usually, first, the pectoralis major and minor; then the trapezius, latissimus dorsi, the serratus magnus, rhomboidei, sacro lumbalis, etc. Usually there are spared the sterno-cleido mastoid, levator anguli scapulæ, coraco-brachialis, teres major, teres minor, deltoid, infra-spinatus, supra-spinatus, and the small muscles of the hand. In the lower extremities, which are later affected, the glutei muscles, the quadriceps, and the peronei are the ones chiefly involved. The sartorius and the calf muscles are only atrophied very late in the disease. Fibrillary twitchings are not seen; reaction of degeneration is not present.

(3) **Infantile muscular atrophy, beginning in the facial muscles.** This form of muscular atrophy, that of Landouzy and Déjérine, is probably similar in character to—in fact, the same disease as—Erb's juvenile atrophy, and merely differs, for some unknown reason, in attacking first the muscles of the face. The facial muscles atrophy. The eyes cannot be closed; the atrophy of the muscles about the lips prevents the normal movements of the mouth, so that the patient cannot pucker up his lips for whistling, and talking is imperfect. As the disease progresses, the muscles of the shoulder girdle are affected, and the atrophy may extend to the muscles of the trunk and lower extremity. The appearance of the patient with this form of atrophy is quite characteristic. The face has a listless, sleepy expression; the forehead is without wrinkles; the eyes cannot be completely closed, and on attempting to whistle, or to pucker the lips, or to speak, impairment of the movements of the lip muscles is plainly evident.

(4) **The peroneal type of muscular atrophy** is regarded by some as always due to a neuritis; others, as Charcot and Marie, believe it to be a primary myopathy. This is a disease of childhood or of young adult life, affecting females as well as males, and attacking the peronei and the small muscles of the feet first. Club-foot may result from the atrophy of the muscles.

It may be well here to consider the various causes that may produce muscular atrophy and the means by which we can recognize the various forms. Muscular atrophy may be due to (1) lesions affecting the cortical nuclei, as from some cerebral disease; (2) the medullary nuclei, as in bulbar paralysis; (3) the spinal nuclei, as in acute anterior poliomyelitis or in progressive muscular atrophy; (4) the nerves, as in peripheral neuritis; (5) or the primary trouble may be in the muscles themselves.

Cerebral muscular atrophy, due to some lesion of the cortical trophic centres, that are as yet not definitely located, follows instead of preceding the loss of power. There is usually hemiplegia or monoplegia, and there are usually other cerebral disturbances.

The **muscular atrophy of spinal origin** is to be distinguished from that of primary muscular origin by the fact that the latter is (*a*) juvenile; (*b*) generic or hereditary; (*c*) by the localization of the atrophy, that is, in the muscles of the lower extremities, of the shoulder girdle, of the face, the peroneal muscles, and not in the small muscles of the hand, as in progressive muscular atrophy; (*d*) by the absence of fibrillary twitchings; (*e*) the absence of reaction of degeneration. Nor is there in primary muscular atrophy, spasticity, as in amyotrophic lateral sclerosis.

Neuritic muscular atrophy is to be distinguished from primary muscular atrophy by (*a*) the history of trauma, infectious disease, of alcoholic or lead poisoning; (*b*) no hereditary or family history; (*c*) sensory symptoms have been, and perhaps are, present; (*d*) the distribution of the atrophy rarely conforms to the types of primary muscular atrophy; (*e*) the paralysis is greater than the atrophy warrants; (*f*) there is a peculiar *steppage* gait, which is not present in primary muscular atrophy.

TABES DORSALIS—LOCOMOTOR ATAXIA.

The more recent views of the pathology of locomotor ataxia, making of it a disease not alone of the spinal cord, but of the spinal and cranial nerves, of the nerve nuclei, and even, according to some, of the cerebral cortex or its white substance, making it in short a disease of the general nervous system, enable us the easier to understand the many variations from the common type of this affection.

One may well say there is no typical case where the lesions are so disseminated. Yet so often the disease manifests itself by lightning-like pains in the lower extremities, by loss of the patellar reflex, and by changes in the action of the pupillary muscles, that one must be pardoned for keeping constantly in mind this group of symptoms as "typical" symptoms of locomotor ataxia in its early or preataxic stage, the only period when the diagnosis presents difficulties and the period when the diagnosis is of importance, as at this stage alone can anything be done in the way of cure. After months, or years, the evidences of inco-ordination become prominent, the gait becomes ataxic, the patient sways to and fro when the eyes are shut, there are anomalies of sensation, disturbances of vision, rectal and bladder complications, until finally the patient, if not succumbing to some intercurrent disease, becomes bed-ridden in the paralytic stage. Though not by any means clearly defined in every case, the three stages of tabes (1) preataxic, (2) ataxic, (3) paralytic, are well worth remembering as helping to fix more clearly in mind the ordinary clinical course of the disease.

The symptoms produced by the spinal lesion of the posterior columns—that most constantly found in the affection—are the most common, and in many respects the most valuable from a diagnostic point of view. They have to do with motility, sensibility, and the reflexes.

Subjective disturbances of **sensation** are usually among the early symptoms. Muscular pains compared to those that follow excessive muscular exertion, as after rowing, climbing, riding, are sometimes the precursors of the more severe and characteristic pains dependent on irritation of the posterior roots. These latter pains vary in character, intensity, and situation. They are almost always in the lower extremities or in the small of the back, are dull and boring in character, are often regarded as rheumatic, and may last for a few minutes or for several hours. Again, the pains may be sudden and of most excruciating severity, aptly termed shooting or lightning pains. The attack may last for a few minutes or much longer, but recurrences are common. Some patients expect a siege of these paroxysmal pains every few weeks or months. Rarely ecchymotic spots or cutaneous swellings are developed at the time of these attacks.

Paræsthetic sensations are often met with. Many patients complain of a sense of numbness in the soles of the feet, or they have the sensation of walking on straw or felt. Hyperæsthetic areas may be found as well as spots of anæsthesia. The perineum may show diminished sensibility; the anæsthesia of the rectum, of the vagina, and the urethra produce unconscious defecation, pleasureless coitus, etc. The temperature sense and the muscular-sense may be perverted. Two pin points widely separated may be felt as one, or one point pressed against the skin is felt as two (polyæsthesia). The alterations of sensation in tabes are most complex, most notably in the lower extremities. The girdle sensation is occasionally noted. Delayed sensation is common. The prick of a pin in the calf of the leg may not be felt for many seconds.

Motor disturbances depend chiefly upon inco-ordination. Muscular weakness is not a prominent feature of tabes until late. The tabetic learns to watch his feet, using the eyes as the guide rather than the muscular and other senses. Without the eyes as guide the gait is uncertain. Sooner or later, even with the aid of sight, he has a peculiar "tabetic gait," throwing the legs out loosely, letting the foot come down with a stamp, the heels first. Later he walks with a cane or two canes, and finally is unable to walk even with these aids, and becomes practically, instead of a locomotor ataxic, a locomotor paralytic.

Romberg's symptom—inability to stand erect without swaying when the feet are brought close together, and the eyes are shut—is present in almost every case, and is a valuable sign, though not confined to tabes. Ataxia of the upper extremities is rarer than of the lower, and is usually later in making its appearance. Evidences of motor irritation, as muscular twitchings and involuntary movements, are rare.

The loss of the patellar reflexes is rightly looked upon as of great value in attempting to establish a diagnosis of tabes. While it is normally absent in a very small percentage of persons, and while other diseases than tabes may cause its disappearance, *e. g.*, neuritis, poliomyelitis, diabetes, alcoholism, it is nevertheless true that, taken in conjunction with the lightning pains, the pupillary changes, and Romberg's symptom, it is of extreme value from a diagnostic point of view. In a few cases the patellar reflex is preserved until late, or even until the end of life. But these are the exceptions. In these cases the root zone is free from pathological change.

The reflex mechanisms of the **bladder, rectum,** and **sexual organs** are usually disturbed in tabes, though often not until late. Among the bladder disturbances may be mentioned a difficulty in the extrusion of the urine, retention of urine, or later a paresis of the sphincter, making it difficult for the patient to hold the urine. Constipation is common in tabes, and at times the rectum is anæsthetic, and the patient is unable to tell whether the bowels move or not. In other cases, the patient has a sensation as if a foreign body were in the rectum when there is none. This produces very annoying tenesmus. Late in the disease the sexual appetite is markedly diminished. There are certain disturbances of the bladder, rectum, and sexual organs that are known as **visceral crises.** They are among the rarer crises of locomotor ataxia. These are pains in the region of the bladder, of the rectum, and of the sexual organs, notably the clitoris. These pains occur paroxysmally, as do those more commonly met with, the gastric crises.

Certain disturbances and anatomical changes are met with in tabes that are usually attributed to alteration of vasomotor and trophic function. Among these may be mentioned hyperidrosis, deformities, loss of the nails and teeth, the perforating ulcer of the foot, and peculiar joint affections, arthropathies or Charcot's joints. These joints are usually painless, become swollen rapidly, even in the course of a single night, show crepitation, even dislocations from the great destruction of bone tissue that takes place quite rapidly. The bones may be fractured readily, as they are extremely fragile. The exact pathology of a Charcot joint is not yet clearly understood.

Not a few of the symptoms of locomotor ataxia are due to the involvement of the **brain** and of the **cranial nerves**, or of the nerve centres. The optic nerve may show atrophy, and there is not infrequently a narrowing of the visual field. Total blindness may be the result of optic atrophy. Paralysis of the ocular muscles may cause disturbance in vision, notably diplopia. Examination of the pupils is of great value, as they are frequently contracted, often of unequal size, and they show a loss of reaction to light, while they may accommodate for distance. To the pupil thus reacting on accommodation, but not to light, the name **Argyll-Robertson pupil** has been applied. Hemicrania is occasionally met with. The auditory nerve is rarely affected.

Disturbances in the vagus nerve probably explain the pharyngeal and gastric crises of tabes. The **gastric crises** are very common, and may be among the early manifestations of the disease. There are usually gastralgia, retching, and vomiting, the ejected matter, though not always, being strongly acid. Patients may suffer weekly, monthly, or at much longer intervals from repetitions of these gastric crises. Laryngeal crises are less frequently met with. They appear usually quite suddenly, and resemble much an attack of spasmodic croup. The hypoglossal nerve is sometimes affected, and as a result there is hemiatrophy of the tongue.

Among the cerebral disturbances that occur in the course of locomotor ataxia must be mentioned vertigo and certain mental disturbances, such as melancholia and even dementia.

If, now, we group together the most common symptoms of tabes, we should say that of the greatest importance from a diagnostic point of view are the lightning pains, the loss of the patellar reflex, the alterations in the pupil. Romberg's symptom, the paræsthesiæ of the lower extremities, the gastric and other crises are of great aid in making a diagnosis.

The disease very rarely runs an acute course, and oftentimes extends over many years. The patient's attention is frequently entirely taken up with the disturbances of the stomach, of the rectum, bladder, or the eyes, so that the subjective symptoms do not call the attention of the physician at once to the spinal cord as the possible seat of the primary trouble. It is of the greatest importance, therefore, that we should in all cases of unexplained paroxysmal vomiting examine as to the possibility of the existence of tabes. So, too, in pains referred to the bladder or to the rectum, where no local anatomical cause explains the pain. And particularly should the physician be on his guard against mistaking the pains of locomotor ataxia for rheumatism, for which they are usually taken by the patient. As has been said above, the early diagnosis is difficult, but it is also an important diagnosis, as in the early stage alone can any hope of improvement be offered.

The combinations of symptoms met with in the early stages of tabes can be, and often are, most varied. Oppenheim mentions as among those most commonly met with the following:

a. Lancinating pains, loss of patellar reflex, Argyll-Robertson pupil.

b. Weakness of bladder, loss of patellar reflex, girdle feeling.

c. Optic atrophy, loss of patellar reflex, or girdle sensation, with corresponding hyperæsthesia, analgesia.

d. Optic atrophy, lancinating pains, impotence.

e. Attacks of vomiting, loss of patellar or pupillary reflex.

f. Arthropathies, analgesia, loss of patellar or pupillary reflex.

g. Paralysis of vocal cords (with or without attacks of coughing), loss of patellar reflex, Argyll-Robertson pupil.

h. Paralysis of ocular muscles, girdle sensation, analgesia, etc.

ATAXIC PARAPLEGIA.

Gowers has given the name ataxic paraplegia to a combined sclerosis of the lateral and posterior columns. The symptoms vary in different cases according as the sclerotic process is the more advanced in the one or the other portion of the cord. Pain is rarely prominent, but there is complaint of a tired feeling in the legs. Increased reflexes and muscular rigidity are present as in lateral sclerosis. Late in the course of the disease the reflexes may be diminished or lost. Inco-ordination is prominent; the gait is uncertain and swaying. Romberg's symptom may be well marked. The Argyll-Robertson pupil cannot be detected.

From tabes dorsalis the disease is readily differentiated by the absence of sensory symptoms, the increase of reflexes, the muscular rigidity, the absence of ocular symptoms. The inco-ordination, with the swaying, uncertain gait; the late disappearance of reflexes distinguish it from lateral sclerosis.

FRIEDREICH'S ATAXIA.

Friedreich's ataxia is a disease often attacking several children in the same family. In many cases an hereditary tendency to this form of nervous disease can be made out. The lesion is situated in the posterior and lateral columns, and often involves as well the cerebellar tracts and the periphery of the cord.

The first symptoms of inco-ordination are manifested in childhood. The child is noticed to walk somewhat unsteadily, to stumble easily, and finally develops an ataxic gait that resembles somewhat that of a drunken man. The gait is swaying or staggering, not the stamping gait of tabes. Ataxia of the arms usually develops early, but always after the development of ataxia in the lower extremities. The movements of the arm may be quite irregular, even choreiform in character. The reflexes are lost early. In marked contrast to locomotor ataxia, sensory symptoms are absent, as are also the ocular changes of tabes dorsalis. Nystagmus is present in the majority of cases, with a disturbance of speech through motor inefficiency of the lips and tongue. The bladder and rectum are intact. Deformities of the feet, and even of the spinal column, may develop late in the disease.

The main points to be considered in the diagnosis are the early age at which the disease appears, the occurrence of the affection in other members of the family, the ataxia of the legs, with the peculiar swaying or staggering gait, the ataxia of the lips and tongue, leading to alteration of speech, the nystagmus. The absence of sensory symptoms and of the Argyll-Robertson pupil also furnish aid in making up a diagnosis.

PRESSURE PARALYSES OF THE CORD.

Where the spinal cord is compressed with sufficient force, marked disturbances in the function of the cord below the point of pressure are manifested, usually in the shape of paralysis. To this form of paralysis the name of *pressure paralysis* has been given, or sometimes the not so strictly accurate name, *pressure myelitis.* The cord is usually at first softened, but later may become sclerotic. In some cases, where marked paralytic symptoms have been present during life, the post-mortem examination has shown very few gross or even microscopic changes.

The commonest cause of pressure paralysis is tubercular spondylitis, with dislocation of a vertebra, or a tubercular abscess, making pressure upon the dura, and in that way upon the cord. Among the rarer causes must be mentioned malignant growth of the vertebræ, or of the meninges, gummata, aneurisms, exostoses. A dislocated or fractured vertebra may also, unless replaced, cause pressure.

Sensory symptoms are usually pronounced, though sensibility may be preserved long after there is complete motor paralysis. At the site of the lesion there is often pain, tenderness upon pressure or upon motion of the spine. Neuralgiform pains shoot down the course of the spinal nerves, may be very severe; tingling and other paræsthetic sensations are frequently complained of; the muscles below the portion of the cord affected become somewhat weak, stiff, spastic. Finally complete paralysis may develop. If the cervical cord is affected the arms are paralyzed; the legs in these cases may be but slightly affected, or completely paralyzed. As in transverse myelitis, the reflexes are exaggerated, unless the lumbar cord be involved, when they are lost. As in lateral sclerosis, there may be a spastic condition; ankle clonus may be marked. Herpes, urticaria, dry skin and nails, bedsores, are among the trophic disturbances that are frequently seen. The rectum and bladder are involved. Constipation is the rule, though later there may be incontinence of feces. At first, there is retention of urine, later incontinence, not infrequently cystitis.

The diagnosis of pressure paralysis is made first upon the recognition of the primary disease. This, in the vast majority of cases, is tuberculosis of a vertebra; we therefore inquire as to tubercular history, search for tender spots over the spine, seek for Pott's boss or other deformity of the spinal column. Secondly, the diagnosis is made upon the (*a*) sensory disturbances—tinglings, neuralgic pains, blunted sensation; (*b*) motor disturbance—paresis, spasticity, paralysis; (*c*) the exaggerated reflexes; (*d*) the paralysis of bladder and rectum; and (*e*) trophic disturbances, as bedsores, urticaria, etc. Where cancer is the primary disease, we usually find the patient over forty years of age. The local deformity is rarely so great as in tuberculosis. There are usually enlarged glands, open to inspection or palpation. We may detect a cancerous nodule in other parts of the body, as in the stomach, the uterus, the mammæ; there is a cancerous cachexia. In carcinoma of the vertebræ, too, pain is usually extremely severe, so that to this form of pressure paralysis the name **paraplegia dolorosa** has been sometimes applied.

CAVITIES AND FISSURES OF THE SPINAL CORD.

Dilatation of the central canal of the spinal cord produces a condition known as hydromyelia. Cavities and fissures situated outside the canal of the cord constitute syringomyelia, generally regarded as a gliosis. In syringomyelia the cavities are usually close to the centre, and usually involve the substance of the posterior columns. Longitudinally they may be limited or may extend the greater portion of the length of the cord.

Slight degrees of hydromyelia may be symptomless. The clinical picture of syringomyelia will differ with the size and situation of the cavities. If, for instance, the posterior columns and cornua are mainly affected, a disease picture resembling somewhat tabes may be present. If the lateral columns are mainly involved, the disease may resemble lateral sclerosis. The diagnosis in these cases may be impossible, though the disease may be suspected by the atypical picture presented.

The most characteristic form of syringomyelia is that in which the cavities form primarily in the cervical cord. Here quite a definite clinical course is run. Muscular atrophy appears in the upper extremities, often as in progressive spinal muscular atrophy, in the small muscles of the hand, then in the forearm, the deltoid, etc. Contractures may appear. The extremity becomes weak, sensibility is disturbed, in that there is loss of the temperature-sense and of the pain-sense, while the tactile sensibility remains normal. Trophic disturbances may or may not be present. The picture resembles quite closely that of amyotrophic paralysis, but the diagnosis in typical cases, where there is progressive muscular atrophy with retention of tactile sensation but loss of sensations of pain and of temperature, is not difficult.

Morvan's disease is a chronic affection characterized by neuralgic pains, anæsthesia of the skin, and painless felons of the ends of the fingers, gradually leading to necrosis. Autopsy in some of these cases has revealed the presence of cavities in the cord In this disease there is tactile as well as thermal anæsthesia. The disease is possibly a syringomyelia associated with neuritis.

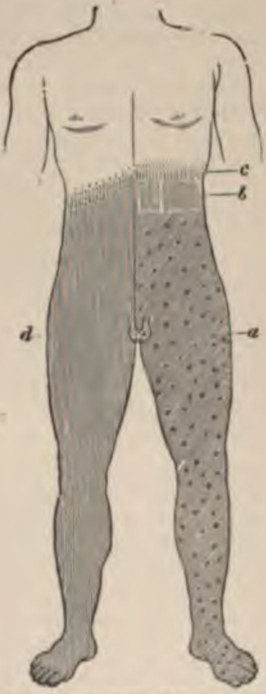
TUMORS OF THE CORD.

Sarcoma, fibroma, gumma, tubercle, as well as other rarer tumor growths, have been met with in the substance of the cord. The clinical picture will, of course, vary with the rapidity of growth, the size of the tumor, as well as its location. The symptoms are those of a pressure paralysis. It is remarkable for how long a time the pressure is in some cases well borne. If the meninges and the nerve roots are involved, the pain may be very severe, radiating along the course of the spinal nerves. The girdle sensation may be present. Areas of hyperæsthesia or of anæsthesia may be noted. Later, motor paralysis occurs. This may at first, when but one-half of the cord is affected by pressure, be of the Brown-Séquard type. One of the most characteristic features of tumors of the cord is the **asymmetry** of the symptoms early met with in the course of the disease. It is quite characteristic, also, that there may be marked **remissions**, the patient being, at least symptomatically, now better and now worse. The clinical picture often resembles very closely myelitis, though myelitis is usually more sudden in its onset, attended by less pain, is more symmetrical, very rarely unilateral, and does not exhibit the marked remissions of tumor of the cord. The prognosis in the case of tumor is uniformly bad, except in operable cases.

BROWN-SÉQUARD'S PARALYSIS OR UNILATERAL LESION OF THE CORD.

Stabs from a knife or sword, or pressure of a tumor upon the cord, may disturb the function of one-half of the spinal cord. As a result of this there is motor paralysis on the same side of the body as the lesion in the cord, and a sensory paralysis on the other side of the body. This is explained by the fact that the motor fibres are continuous upward to the medulla without crossing, on the same side on which they enter the spinal cord, while the sensory fibres cross to the opposite half of the spinal cord and pass upward, probably in the gray matter. This is made clear by the accompanying figure. On the side that is paralyzed there is usually hyperæsthesia, but the muscular sense is absent. Above the hyperæsthetic area is an anæsthetic area, and above this, at times, a hyperæsthetic zone. The reflexes are increased on the paralyzed side.

FIG 72.



Schematic representation of the chief symptoms of unilateral lesion of the left dorsal cord. The oblique shading signifies motor and vasomotor paralysis; the vertical shading signifies cutaneous anæsthesia; the dots signify cutaneous hyperæsthesia. (ERB.)

The temperature of the skin may be slightly elevated. On the side that is anæsthetic the muscular sense is normal, as is motion. The bladder and rectum are usually affected. Neuralgic pains, and muscular atrophy may be present.

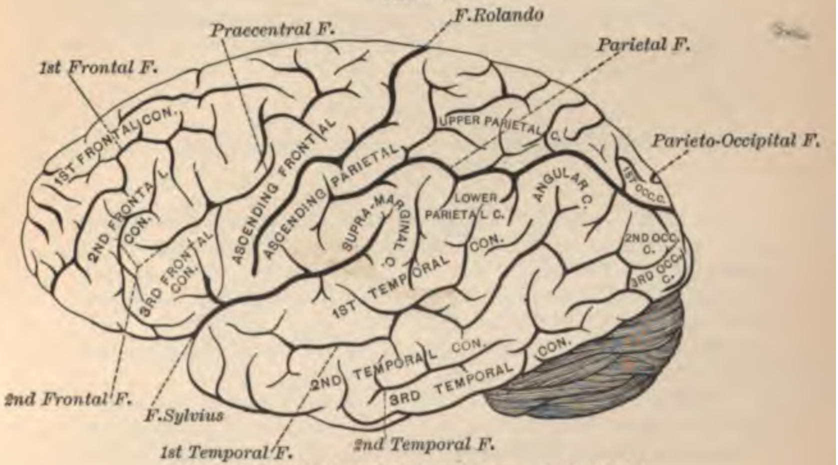
THE BRAIN AND ITS COVERINGS.

CEREBRAL LOCALIZATION.

Certain cerebral convolutions are known to govern muscular movements, others have to do with sensation, others with psychic manifestations, and still others with sight, hearing, smell, touch.

Motor Centres. The region about the fissure of Rolando is the motor region. The posterior portion of the frontal convolutions, the ascending frontal and the ascending parietal convolutions (*gyrus centralis anterior* and *gyrus centralis posterior*), a portion of the parietal lobule, and of the paracentral lobule (*i. e.*, the portion of the marginal convolution just anterior to the quadrate or pre-cuneate lobule) make up this area.

FIG. 73.



Convolutions of left cerebral hemisphere. (L. C. GRAY.)

The lowest portion of this region, that next the Sylvian fissure, includes the centres of the face, the tongue, the larynx. Just above this area is the region for the arm, the centres for the thumb, fingers, elbow, shoulder, being in the order named from below upward. Above the arm centre, and occupying the upper quarter of the Rolandic area, and bordering on the longitudinal fissure, are the centres for the leg, the centre for the hip being in front of the fissure of Ronaldo, and the centre for the smaller toes behind it. The trunk muscles are governed by a centre in the paracentral lobule of the marginal gyrus.

FIG. 74.

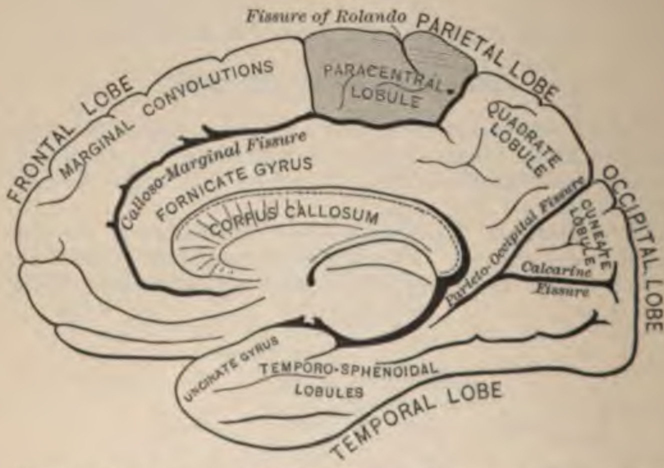


Diagram of the median surface of the cerebrum. B – corpus callosum. (After ECKER-STRÜMPELL.)

Fibres from these motor centres pass by way of the motor or pyramidal tract to the peripheral nerves and the muscles. This tract leads from the cortex through the corona radiata to the internal capsule. The fibres, here compactly aggregated, are so arranged that those for the leg are situated posteriorly; most anteriorly, at the knee of the capsule, are those for the face, tongue, eyes, and between the arm fibres. (*Vide* Fig. 77.)

From the internal capsule the fibres pass in succession through the crus, the pons, the medulla. Most of the fibres here descussate and pass down the opposite side of the spinal column as the crossed pyramidal tract. A small number of fibres, variously estimated at from one-tenth to one-fourth the entire number, form Turck's column or the anterior pyramidal tract. Entering the gray matter of the cord, joining the anterior cornual nerve-cells, the fibres are continued from the anterior horns as peripheral motor nerves.

By a **secondary descending degeneration** is meant a degeneration that occurs in the fibres along the motor tract just described, below any point beyond which communication with the cortical cells is cut off. Thus a cortical lesion causing destruction of cortical cells produces descending degeneration throughout the entire motor tract as far as the cells of the anterior horns. A lesion of the crossed pyramidal tract in the mid-dorsal region produces a descending degeneration in the tract below this point. The cells of the anterior cornua, believed to have trophic functions, still remain intact and serve as a barrier to prevent further extension of the degenerative process.

A lesion of one of these motor centres that destroys a centre, or at least destroys its functions—a destructive lesion—produces paralysis in the group of muscles (on the opposite side, of course) supplied by the centre. The paralysis is accompanied by exaggerated reflexes, increased muscular tension, but not by degenerative atrophy nor the reaction of degeneration.

FIG. 75.

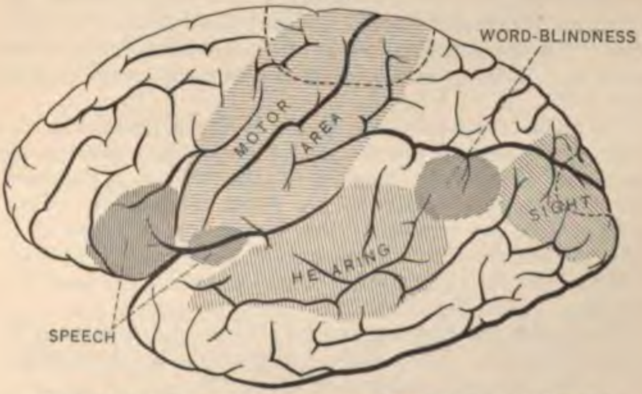


Diagram showing localization of centres in the cortex. [L. C. GRAY.]

If all the motor centres of one cerebral hemisphere are destroyed, hemiplegia is the result.

Irritative lesions may produce localized spasms, often of a convulsive nature, in single muscles or groups of muscles—a Jacksonian epilepsy. The progress of growth in a tumor of the cortex can often be followed, as at first symptoms of irritation may be present, as, for instance, localized convulsions in facial muscles, often with abnormal sensations. Then, as the tumor grows, irritative symptoms may be manifested in the fingers, the arm, the shoulder, and later the leg. By the time the irritation of the leg centre is manifested, there may be a complete paralysis of the muscles of the face area and of the arm area; in other words, the irritative lesion has become a destructive lesion. The same change is sometimes seen in cases of cerebral meningitis where, as the exudate becomes great in amount, the symptoms of a destructive lesion (pressure of exudate) replace those of an irritative one.

The sensory centres have been variously located in the posterior half of the motor area, in the gyrus fornicatus, the gyrus hippocampi, etc. Exact knowledge on this point is still lacking.

The psychic centres are also not definitely located, though in many cases lesions of the superior and middle frontal convolutions have had few symptoms save those of a psychic character. On the contrary, lesions of the frontal lobes have been found *post-mortem*, where no mental symptoms had been noted during life.

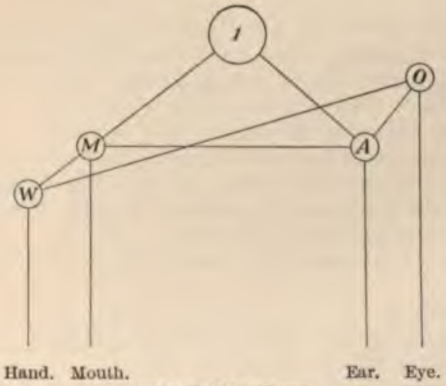
The sight centre of the cortex is in the occipital lobe. A destructive lesion of one occipital lobe produces hemianopia, the retinal fibres of the same side of either eye being paralyzed, and therefore the opposite field of vision being blind. This is made clear if we remember the decussation of fibres at the optic chiasm, and that the left half of the retina receives rays of light from the right field of vision and the right half from the left. (*Vide* Fig. 75.)

The centre for hearing is probably in the first temporo-sphenoidal lobe. The centre for smell is in the tempero-sphenoidal lobe and in the uncinate gyrus.

Aphasia. The centre for speech is situated in the left third frontal convolution, Broca's convolution. In left-handed people it is usually in the right hemisphere. A destructive lesion of this centre renders the patient unable to put his ideas into articulate language. He hears perfectly, recognizes objects seen, understands questions, tries to speak, knows the words he wishes to use, but is unable to articulate them. He is able to make his wants known by nodding the head, by pointing, by grunts of approval or of disapproval; but the movements of lips, tongue and larynx necessary to produce words he cannot control. He is as a child who has not learned to talk. This is **motor** or **ataxic aphasia.** Incomplete motor aphasia may show as an inability to use all words correctly. The patient may stumble over syllables; says, for example, *them* for *then; anagolous* for *analogous; though* for *thorough*, etc. Or he is able only to repeat one word or a single expression, which he uses in answer to all questions. One of my patients could use but an oath and no other word.

If the patient is able to articulate, but cannot remember the right word, he is said to have amnesic aphasia. He is like a stranger in a foreign land, ignorant of the language spoken. He recognizes objects, knows their uses, but cannot speak the words necessary to make himself understood. If the word is pronounced, he has the motor power necessary to repeat it. But he has forgotten the word he once knew, and try as he may he cannot recall it. Where there is partial amnesic aphasia, only certain words may be forgotten, and the patient gets along on a limited vocabulary.

FIG. 76.



Lichtheim's schema.

A modification of amnesic aphasia known as word-deafness is that form in which the patient hears the word, but the sound fails to call up the appropriate mental image. He has forgotten what the word signifies. He is as the foreigner who hears the strange language, but for whom it has no meaning. Word-deafness—and probably amnesic aphasia proper—is due to a lesion in the first left temporal convolution.

Word-blindness, where the sight of an object fails to call up the appropriate mental image, is supposed to have its seat in the angular gyrus.

Closely allied to aphasia is agraphia—inability to write; alexia, inability to read.

By a study of Lichtheim's schema, here given, the mode of production of aphasia and its varieties may be more clearly understood.

The location of the lesion in different forms of aphasia and allied disorders is as follows:

1. **Apraxia**—mind-blindness, the loss of power of recognizing the nature, use, and relations of objects—lesion at *I* (or $I + A + O$.
2. **Word-deafness**—*A*—first left temporal.
3. **Word-blindness**—*O*—angular and supra-marginal.
4. **Paraphasia**—Tracts *AM* and *OM*. This is an aphasia partially motor, partially sensory.
5. **Motor Aphasia**—*M*—Broca's convolution.

Where the difficulty is in writing instead of in talking the centre, *W*, is involved instead of *M*. The question as to whether there is a separate centre for writing, independent of the speech centre, is yet undecided.

In cases of aphasia the following tests should be made (Osler):

The power of

1. Recognizing nature, use, relations of objects. (**Apraxia.**)
2. Recalling names of familiar objects.
3. Understanding spoken words. (Word-deafness.)
4. Understanding printed or written language.
5. Understanding and appreciating musical tones.
6. Voluntary speech—whether or not words are misplaced.
7. Reading aloud and of understanding what is read.
8. Writing voluntarily and of understanding what is written.
9. Copying.
10. Writing at dictation.
11. Repeating words.

Centrum Ovale. The white substance of the hemisphere contains commissural fibres uniting the corresponding white substance of the two hemispheres, as well as fibres uniting adjacent convolutions. Lesions of the centrum ovale, destroying these fibres, are not recognizable as such during life. There are, in the centrum ovale, the fibres converging from the cortical centres to the internal capsule. If these fibres are destroyed, motor paralysis follows just as though the cortical centre from which the fibres sprang had been destroyed. In this way hemiplegia or monoplegia may occur. If the fibres from the occipital lobe are cut off, hemianopia results; from the third left frontal, motor aphasia; from the third left temporal, word-deafness, etc. Lesions of the frontal white matter may be symptomless. The diagnosis of all lesions of the centrum ovale is extremely difficult or impossible.

Internal Capsule. In the internal capsule the motor fibres are so closely compacted that a slight lesion, as by the tearing of the capsule or by the pressure of a neighboring hemorrhage, may produce hemiplegia of the opposite side; if the posterior part be also involved, the sensory fibres are cut off, and hemianæsthesia results. The special senses are usually disturbed by the latter lesion.

FIG. 77.

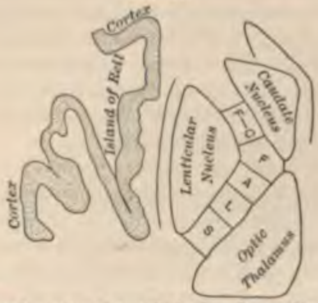


Diagram of horizontal section through the basal ganglia and internal capsule (left side), showing the position of the chief tracts in the internal capsule. The region of the capsule marked by the letters L, A, F is occupied by motor fibres; L, corresponds to the leg-fibres; A, to the arm-fibres; F, to the face-fibres (including fibres to face muscles and tongue). The region, F-C, contains the fronto-cerebellar tract (intellectual tract). The region marked S contains the general sensory tract from the opposite side, and the fibres from the optic and olfactory nerves of the opposite side, sometimes called the "sensory crossway" (HERTER.)

Crura Cerebri. A lesion here produces results similar to those in the internal capsule. The motor oculi nerve (third) is often involved in this lesion. Paralysis of ocular muscles on the same side as the lesion would therefore result, with hemiplegia of the opposite side.

Pons. A destructive lesion here produces hemiplegia of the opposite side, and usually a paralysis of the facial nerve on the same side, *i. e.*, a crossed paralysis. If the middle peduncle of the cerebellum be involved at the same time, either directly or by pressure, the patient may tend when walking, to fall forward toward the side of the lesion.

Optic Thalamus, etc. But little is known of the localization of lesions of the central ganglia, the thalamus, the caudate and lenticular nuclei. A destructive lesion, however, of the hinder part of the optic thalamus will produce hemianopia of the opposite side. Destruction of the anterior pair of the corpora quadrigemina produces blindness.

Cerebellum. Disease of the cerebellar hemispheres may produce few or no symptoms. If, however, the middle lobe is affected there is generally ataxia, vertigo, headache, vomiting. The ataxia is shown when the patient attempts to walk, in a reeling, staggering gait, often with a tendency to pitch forward or to one side. The upper extremities are not ataxic. Vertigo is noticed in the erect position, but not when the patient is recumbent. Occipital or frontal headache is common, and vomiting is usually present.

CEREBRAL MENINGITIS.

The pathological division of meningitis into the purulent, tubercular, and epidemic cerebro-spinal forms is fairly well preserved clinically. Acute and chronic forms of each group are also recognized.

Acute Purulent Meningitis.

In this form, as in the tubercular, it is common to speak of three stages: (1) The stage of irritation with headache, delirium, vomiting; (2) the stage of compression with slow pulse, stupor, and paresis; (3) the stage of paralysis with rapid pulse, paralysis, coma. In some cases this succession of stages can be fairly well made out. In many, however, there is no clear transition from one stage to another, and often the phenomena of irritation, compression, and paralysis are presented at one and the same time.

In attempting to diagnose a meningitis the cause should always be sought for. An injury to the head, a suppurating middle ear, a suppurating stump left after amputation of the eye, a pus focus in some distant part of the body, *e. g.*, in the liver, or the presence of some infectious disease, as pneumonia, erysipelas, scarlet fever, may clear up the case wonderfully by furnishing us a cause for the infection of the meninges, either by direct extension or by the avenue of the blood channels (hæmatogenic, as in the diplococcal meningitis of pneumonia).

One of the most prominent, early, and persistent symptoms is headache, for which no relief is obtained save by opiates. And even when under opiates or in the stupor of compression, the patient complains of the head or keeps putting the hand to the head whenever he is disturbed. Mental symptoms vary from slight wandering or even marked delirium at first, to stupor and deep coma toward the last. Vomiting, often projectile and having no connection with the taking of food into the stomach, is common.

On examining such a patient, there is found a slow pulse, that, as the disease progresses, becomes more irritable and variable, and later rapid and weak. The temperature is elevated slightly or to a high point. Just before death hyperpyrexia has been often noted. The respirations are sometimes hurried, and Cheyne-Stokes breathing is often seen as the disease advances toward a fatal termination. The pupils are often contracted, perhaps unequally. There is developed sooner or later, a rigidity of the neck, and there may be marked retraction. The skin may be hyperæsthetic. The condition of the reflexes is of slight value in diagnosis. The basal meninges being so often the site of the inflammatory process, there are disturbances in the functions of the cranial nerves. Strabismus and nystagmus may appear. Disturbances of sight and of hearing are not uncommon. There may be choked disk. The facial nerve is frequently paretic or paralyzed. With cortical involvement there may be muscular twitchings or rigidity, and later complete paralysis. General convulsions may occur. Aphasia has been observed. Death preceded by coma, rapid pulse, and high temperature, is the common result.

Epidemic Cerebro-spinal Meningitis,

which has already been considered, presents symptoms varying but slightly from those just detailed. There are, however, usually other cases of the disease about. A chill or a convulsion is common at the beginning. Herpes, splenic tumor, the skin lesions, the early and marked retraction of the neck, the tenderness of the spine sometimes present, may enable us to separate this form of meningitis from the simple purulent and tubercular forms. Recovery, complete, or with some defective cerebration, is common after the epidemic form.

Tubercular Meningitis

is commonly basilar, and met with oftenest in children. A primary focus cannot always be discovered, being often tubercular bronchial glands. It may be a part of a general miliary tuberculosis.

In its onset and course it may be as sudden and rapid as the suppurative form, and may perfectly resemble it. As a rule, however, the onset is gradual. The child complains at times of the head, is peevish and irritable, sleeps poorly; while at play or even during the night will give a sudden, sharp cry—the cephalic cry. Soon fever comes on, cerebral vomiting occurs, and delirium, strabismus, unequal pupils, blindness, retracted neck, slow pulse, give the unmistakable evidences of involvement of the meninges. Convulsions, stupor, rapid pulse, are met with, as in the suppurative form. Tubercles are sometimes seen in the choroid.

Subacute and chronic forms of all varieties of meningitis are met with. The tubercular and the epidemic forms may last for weeks.

Differential Diagnosis. The tubercular form is differentiated from the purulent, where this is possible, by the discovery of a tubercular focus in some part of the body, the tubercular hereditary history, by the slower onset, the lower fever, the slower course, the detection of tubercles in the choroid. Leucocytosis is less marked, or absent, in the tubercular form.

In the subacute forms the resemblance of meningitis to **typhoid fever** is very striking. There is the same initial anorexia, malaise, headache, sleeplessness, chilliness, fever. The stupor, the dry tongue, the muscular twitchings, the emaciation, the muttering delirium, may well deceive, as the simulation may be almost perfect. In meningitis, however, if the opportunity of watching the case long enough is afforded, there develop the paralyses of ocular muscles, the rigidity of the neck, the paralysis. Hyperæsthesia, retracted abdomen, optic neuritis, constipation, may help in the diagnosis of meningitis; while blunting of cutaneous sensitiveness, tympany, enlarged spleen, rose-spots speak for typhoid. Ehrlich's reaction, if absent, is in favor of meningitis, *i. e.*, helps exclude *typhoid*.

With **pneumonia** it may be impossible to determine whether meningitis is present or not, until paralysis of the eye muscles or of the facial nerve is seen.

Uræmic convulsions or coma can generally be recognized by the previous history of the case, by the urinary findings, the œdema, the evidence of cardiac hypertrophy, the absence of rigidity of the neck, and of ocular paralyses.

SUNSTROKE.

Exposure to extreme heat, whether to the direct rays of the sun or not is immaterial, particularly when the atmosphere is moist, frequently results in what is known as sunstroke or thermic fever.

Two forms may be recognized. The first is **heat prostration**, in which the patient feels faint, becomes nauseated, and suffers from great muscular and general weakness. The pulse is rapid and feeble, the body is cool, and recovery usually occurs in a few days. After-effects may, however, be present in these cases in the shape of great sensitiveness to heat, or some mental derangement, or severe and persistent headache.

The second variety or the **thermic fever** is at times preceded by prodromal symptoms, dizziness, blinding headache, and ringing in the ears. The temperature rises very rapidly to 105° or even 110°. The patient falls unconscious, and in some cases expires within a few minutes. The pulse is slow and full at first, but toward the last may be quick and feeble. Respiration is rapid, at times stertorous, and may be irregular. Vomiting and involuntary urination and defecation are common. There is great restlessness, and the limbs are in almost constant motion; there may be convulsions or coma. Death occurs unless the temperature is quite rapidly reduced.

A knowledge of exposure to extreme heat, the temperature so unusually high, the vomiting, the jactitation, the involuntary discharges, the stupor or coma, render the diagnosis easy. The disease might be confounded with meningitis, with acute alcoholism, or with epilepsy, but careful examination will exclude these conditions.

CEREBRAL ANÆMIA.

Acute cerebral anæmia may be caused by fright, fatigue, lack of food, great worry or mental anxiety. When extreme, the ordinary symptoms of a fainting spell are manifested.

The patient becomes dizzy and has ringing in the ears; spots are seen before the eyes, the countenance becomes pale, there may be nausea or vomiting, the pulse grows rapid and feeble, and the patient falls unconscious. In chronic cerebral anæmia, as in chlorosis and pernicious anæmia, slight exertion may produce many of these symptoms, as dizziness, ringing in the ears, spots before the eyes, etc., as described under the head of chlorosis and anæmia.

The unconsciousness of a faint is usually recognized without much difficulty. A history of the case gives us a clew as to what is the matter. The lack of convulsions, the pallor of the countenance, the feeble pulse, the deep inspiration on sprinkling cold water over the face or causing the patient to inhale the fumes of ammonia, usually enable us readily to make a diagnosis.

CEREBRAL HYPERÆMIA.

While not a separate disease, cerebral hyperæmia sometimes manifests itself by fairly well-marked symptoms. The face becomes reddened, the carotids pulsate, the head feels warm and full and aches. At times there are hyperæsthesia and nausea. The patient may become greatly excited or even maniacal. He may later be drowsy or even stupid, and the picture may resemble that of a very mild apoplectic shock.

CEREBRAL HEMORRHAGE.

The most frequent cause of cerebral hemorrhage being a diseased condition of the bloodvessels of the brain, it is met with oftenest in adults or the old, in syphilitics, alcoholics, the gouty, *i. e.*, the victims of arterio-sclerosis. Causes that increase the intra-vascular pressure, *e. g.*, chronic nephritis, cardiac hypertrophy, heavy lifting, and violent excitement favor the rupture of these diseased vessels, whose weakened walls have in many instances yielded in miliary aneurismal dilatations. In diseases attended by the hemorrhagic diathesis, *e. g.*, in scurvy, purpura, leukæmia, pernicious anæmia, smallpox, etc., hemorrhage into the brain may occur. Short-necked, fleshy, full-blooded, perhaps wheezy and asthmatic individuals are looked upon as possessing the "apoplectic habit." Heredity is not to be overlooked as a cause.

Hemorrhage is oftenest met with just outside the internal capsule in the neighborhood of the lateral ventricles. Oftenest, too, the hemorrhage is into the left hemisphere. If motor fibres in the corona radiata or in the internal capsule are lacerated and destroyed by the extravasated blood, an irreparable paralysis results. Pressure upon adjacent motor centres or motor fibres, and particularly the fibres of the internal capsule, may produce indirectly a paralysis that is often transitory.

There is rarely a premonition of the accident. In some cases, however, dizziness, tinnitus, headache, slight speech disturbances may presage an attack; circulatory disturbances or minute hemorrhages probably explain these slight symptoms.

The shock or stroke of apoplexy is commonly sud den. The patient falls to the ground unconscious, breathes stertorously and often slowly. The eyes may be turned toward the lesion in the brain; the pupils are often unequal. Paralysis may be noted in the muscles of one side, though at first in the limp and relaxed extremities, but little inequality in power of movement can be detected. Such attacks may be rapidly fatal. In others the patient remains for hours in a comatose condition. Cheyne-Stokes respiration is not uncommonly noted just before death. The temperature, at first subnormal, usually rises later.

In some cases there is a more gradual onset. The patient has a confused, whirling, or painful sensation in the head, the face becomes flushed, he talks thickly and irrationally, notices a weakness in an extremity, and gradually becomes unconscious. The hemiplegia develops rather gradually.

The outcome in a case of cerebral hemorrhage is always at first uncertain. In most cases there is a gradual return to consciousness and a restoration of function in the muscles whose fibres have been inactive because of pressure. Usually the lower extremities regain the power of movement sooner than the upper. Where fibres have been permanently destroyed, there is a descending degeneration with resulting contractures.

The diagnosis of cerebral hemorrhage usually resolves itself into two problems: (1) The diagnosis of the coma; (2) the diagnosis of the post-comatose condition, *i. e.*, the recognition of the character and location of the cerebral lesion.

The diagnosis of the coma of cerebral hemorrhage is made upon the sudden onset, though in rare cases there are prodromal symptoms; upon the detection of sclerotic bloodvessels, the slow pulse, the unequal pupils, and at times a conjugate deviation. There is also discovered the fact that one side is completely relaxed, or much more so than the opposite side, which is perhaps rigid and resisting. The breathing is stertorous; one cheek is drawn in and out, or more so than the other. The reflexes on the paralyzed side are often absent at first.

The coma of cerebral hemorrhage has to be distinguished from various other varieties of coma. Difficulties are frequently encountered, particularly in differentiating between this form of coma and that attending opium-poisoning, alcoholic narcosis, and uræmia. The following table shows the important points on which the differential diagnosis is based:

Cerebral hemorrhage (apoplexy).	Opium narcosis.	Alcoholic narcosis.	Uræmia.
1. Pupils unequal or dilated.	Pupil contracted.	Pupil contracted or dilated; eyes injected.	Generally dilated; retinitis.
2. Stertorous breathing, puffy breathing, cheek flaps.	Calm, slow breathing.	No stertorous breathing.	Sharp hissing stertor.
3. No odor.	Fetid opium breath	Odor of alcohol.	No odor, unless urinous.
4. Paralysis, hemiplegia.	Gradual loss of muscular power.	Unconsciousness, but not paralysis.	No hemiplegia.
5. Unconsciousness.	Can be momentarily aroused.	Momentarily aroused, especially if ammonia is inhaled.	May or may not be momentarily aroused.
6. Pulse slow and strong or irregular; arteries often atheromatous.	Pulse slow until late.	Pulse frequent and feeble.	Pulse at first strong, later weak and rapid; tension strong; arterio-sclerosis.
7. Coma sudden and deep.	Coma gradual (often vomiting).	Coma gradual.	Coma gradual or sudden.
8. Convulsions late; may be unilateral	No convulsions.	No convulsions.	Preceded by general convulsions, headache, etc.
9. Urine generally negative.	Urine contains morphine.	Urine generally negative.	Urine albuminous.
10. Often apoplectic habit; heart may show hypertrophy.	Countenance and heart negative.	Red face and nose; heart often weak, dilated, myocarditic.	Œdema and pallor, heart hypertrophied.

The coma of diabetes is also to be remembered. Here there is often a peculiar fetid or sweetish odor to the breath; the skin of the patient is usually dry and harsh. There may be boils or carbuncles, frequently a dermatitis about the genitalia. The patient may be extremely emaciated; not infrequently tubercular deposits are found in the lung. The urine contains sugar.

The coma of sepsis usually presents little difficulty if we have a history of the case. In the absence of a history, the temperature, the quality of the pulse, the dry, parched tongue with the foul mouth, often a bedsore, give us a clew to the diagnosis.

Among other forms of coma that must be thought of are the coma from chloroform and ether that have been taken as poisons, where the odor of the breath enables us to make a diagnosis, and the coma from cerebral compression from a tumor or abscess of the brain.

After consciousness has been recovered, the question of diagnosis is as to the nature of the lesion and its location. This is determined almost exclusively by the examination for paralysis. The degree varies in different cases. The paralysis is almost always hemiplegia. When upon the left side and involving Broca's convolution there will be aphasia. The facial muscles are paralyzed, or may be paretic. There will be no wrinkles seen, the patient, when laughing or whistling, moves the sound side and retracts the angle of the mouth much more than on the paralyzed side. The upper division of the facial nerve is not affected in cerebral paralysis. Wrinkling the forehead shows that both sides wrinkle alike. The reason why, in cerebral paralysis involving the facial nerve, the upper division is not affected is not known. In peripheral facial paralysis the upper division as well as the others is affected. The tongue when protruded is pushed by the unaffected genio-hyo-glossus muscle toward the paralyzed side, and articulation is frequently interfered with. The soft palate may hang low on the paralyzed side; the muscles of the trunk, save the trapezius, are rarely perceptibly affected. The extremities are motionless; the reflexes at first may be lost; but later, and always when descending degeneration has occurred, are exaggerated. Often upon the sound side the patellar reflexes may be slightly exaggerated. The cutaneous reflexes are diminished or abolished. Sensation is rarely lost. Paræsthetic sensations are not uncommon at first. Where descending degeneration of the pyramidal tract has occurred there are contractures. Where there are contractures the paralysis is permanent. Occasionally choreic or athetoid movements follow a cerebral hemiplegia. Vasomotor disturbances, as glossy, cold, clammy skin, are frequently present. Atrophy is slight, but there is no reaction of degeneration.

The recovery of motion is usually first noticed in the lower extremities. Patients, even with marked paralysis, are able to walk, swinging the leg out by the action of the psoas muscle. Mentally, the patient, following hemiplegia, may be as bright as usual, though often the mind is somewhat sluggish and the patient rather emotional, laughing and crying upon slight provocation.

CEREBRAL EMBOLISM AND THROMBOSIS.

The clinical phenomena attending cerebral embolism resemble very closely those attending cerebral hemorrhage. There may be the same "shock" as in an ordinary apoplexy, though it is apt to be less severe, and is oftener attended by convulsive movements. The consequences—that is, unconsciousness and paralysis—are practically the same. Aphasia is very common, as the left middle cerebral artery is oftenest occluded.

In attempting to diagnose cerebral embolism great value must be laid upon the detection of the source of the embolus. Oftenest this is found in a deposit on a diseased valve of the heart, or in a peripheral thrombus. The age of the patient is of some value, as embolism is more often found in the young than is hemorrhage. Emboli also may be detected in certain cases in the retina.

Cerebral thrombosis, due usually to chronic endarteritis, as in the syphilitic, is ordinarily much slower in its onset, may be attended by relapses. There is rarely anything like the ordinary apoplectic shock.

CEREBRAL ABSCESS.

Abscess of the brain may be due to (1) traumatism; (2) to extension of inflammation from adjacent parts, oftenest the middle ear or the nasal cavities and the ethmoid cells; (3) to septic emboli carried from some distant primary suppurating focus; and (4) to primary infection of the encephalon.

Following traumatism, as fracture of the skull, the symptoms are usually those of an acute septic trouble, and differ very little, if at all, from those of acute suppurative meningitis. There is severe headache, delirium, often vomiting; there may be convulsions; later, paralysis, death following in a few days or weeks. These same symptoms may attend an acute encephalitis that is idiopathic, *i. e.*, cryptogenetic, and that is seen sometimes during epidemics of cerebro-spinal meningitis, so that these cases can scarcely be distinguished from cases of acute meningitis. The same may be said of many of the embolic abscesses. Here the abscesses are more apt to be multiple and the symptoms masked, or overshadowed, by those of the primary disease. Thus, a patient with ulcerative endocarditis may have minute abscesses of the brain which may be readily overlooked, as the other symptoms of endocarditis are so prominent as to throw the headache, the delirium, and vomiting, into the background.

While suppurative encephalitis from otitis media may be acute and with the symptoms above described, it is ordinarily chronic, or the abscess may remain for a long time latent. When the symptoms of cerebral trouble develop there is headache, vomiting, often irritability of temper, perhaps mental hebetude and apathy, and there may be focal symptoms. There is no other localized cerebral disorder in which the focal symptoms are so often absent, either for a long while or even throughout the illness. These focal symptoms, of course, depend upon the location of the abscess. Occasionally irritative effects in the shape of convulsions may be seen; oftener there is paralysis of one limb or of several limbs; where the cerebellum is involved there will be vomiting, loss of coordination, vertigo, particularly if the middle peduncle be involved. Choked disk is not found so often as in case of cerebral tumors.

In making up a diagnosis of cerebral abscess the greatest importance must be attached to the detection of the etiological factor—traumatism, some primary suppurating focus, as an empyema, ulcerative endocarditis, bronchiectasis, and particularly otitis media, or suppurating disease in the upper nasal cavity. There are present also the general cerebral symptoms—headache, vomiting, vertigo. The diagnosis between this disease and tumor of the brain is sometimes impossible where the suppurating focus cannot be detected. With abscess there is less often choked disk. Fever and a typhoid state argue in favor of abscess, though it must be remembered that many abscesses are not attended by fever, and particularly is this true of latent abscesses in the indifferent regions of the brain, as, for example, in the frontal lobes and the lower temporo-sphenoidal lobes. Abscess causes paralysis of the cranial nerves, as the motores oculi, much less frequently than does tumor.

INFANTILE CEREBRAL PARALYSIS.

This is usually a hemiplegia coming on suddenly in young children, with vomiting, fever, convulsions, stupor, or coma that may last for days. Acute infectious disease, as scarlet fever or measles, may, or may not, have been previously present. The convulsions are usually unilateral. Rarely, as sometimes occurs in the case of infantile spinal paralysis, the initial fever, delirium, convulsion may be unnoticed, and the first evidence of trouble is the hemiplegia that is manifest as the child awakes in the morning.

As the weeks go by, the child usually regains the use of some of the muscles, oftenest those of the lower extremity recovering first. Contractures occur in the muscles permanently paralyzed. There may be, in consequence, quite marked deformities, and particularly where the affected limb does not keep pace in its growth with the healthy one. The reflexes are exaggerated, the reaction of degeneration is absent. Sensation remains unaltered. Often mental defects are a consequence of the cerebral lesion. Idiocy, aphasia, epilepsy, are not uncommon sequelæ.

From fœtal encephalitis or from injury done by the forceps at birth, bilateral injury of the cortex may result, with a paralysis of all four extremities, **diplegia**, or there may be paralysis of the lower alone, spastic paraplegia. There are usually mental defects also as a result. The affected members show no atrophy nor reaction of degeneration. Reflexes are often exaggerated. There is marked spasticity and not infrequently there are choreic or athetoid movements.

The palsy of cerebral origin is to be distinguished from that of spinal origin by (1) its hemiplegic character, though it must be remembered that exceptionally cerebral paralysis is bilateral, and that occasionally infantile spinal paralysis is hemiplegic. 2. The presence of distinct cerebral symptoms, aphasia, mental weakness. 3. The slight degree of atrophy. 4. The absence of the reaction of degeneration. 5. The presence of the reflexes, often exaggerated. 6. Post-hemiplegic athetoid movements are frequently present.

The pathological lesion in these cases is usually hemorrhage, embolic softening, or an inflammatory process (polioencephalitis) akin to that occurring in the anterior horns in acute infantile spinal paralysis.

TUMORS OF THE BRAIN.

The commonest varieties of tumors within the brain are glioma, syphiloma, tubercle, which may be solitary or multiple, sarcoma, secondary carcinoma. Other varieties are comparatively rare.

The symptoms of tumor that may be regarded as most characteristic, but which must really be spoken of as general symptoms, as they are present with abscess and the other diseases, are headache, vomiting, dizziness, choked disk.

The **headache** may be diffuse or localized. In the latter case it may be over the site of the tumor, though this is by no means the rule. Tenderness on pressure over the tumor is sometimes observed. The headache is usually continuous, though subject to exacerbations. **Vomiting** is common and is of the cerebral type, that is, shows no relation to the food ingested, is often attended by a perfectly clean tongue, is obstinate, and sometimes projectile. Where the middle peduncle of the cerebellum or the pons is involved, vomiting may be particularly obstinate. **Choked disk** occurs in a large majority of all cases and is usually double. Dizziness is frequently present, and is especially prominent in tumor of the cerebellum. Mental disturbances may or may not be present. The patient may be dull, stupid, apathetic; may be weak-minded, emotional, or even maniacal. Localized convulsions, that is, Jacksonian epilepsy, may occur, or there may be general convulsions.

The local symptoms of tumor, of course, vary with its situation, and may be either irritative or destructive. The irritative lesions are spasm of a muscle or of a muscle group. These frequently precede a paralysis. In many cases the progress of the disease can be followed, as one group after another is involved, first in spasm, and later in paralysis. The localization of the tumor by these symptoms of spasm and of paralysis is of the utmost importance, as it enables the surgeon in many cases, by operation, to remove the growth. The method of localizing the growth has already been given in the chapter on Cerebral Localization.

The following table by Knapp in Strümpell's *Practice* will be of value for reference:

Prefrontal Region. Marked mental impairment; symptoms of invasion of the central region (Jacksonian epilepsy, aphasia); disturbances of smell.

Central Region. Jacksonian epilepsy; monoplegia; partial anæsthesia; motor aphasia.

Posterior Parietal Region. Word-blindness; disturbance of muscular sense (?); homonymous hemianopsia.

Occipital Region. Homonymous hemianopsia; soul-blindness.

Temporo-Sphenoidal Region. Latent region. Word-deafness; disturbances of taste, smell, and hearing (?).

Corpus Callosum. Latent region. Progressive hemiplegia, often bilateral from invasion.

Optico-striate Region. Hemiplegia; contracture. In posterior part, hemianæsthesia, homonymous hemianopsia, post-paralytic chorea, athetosis.

Crus Cerebri. Crossed paralyses of oculomotor nerve and limbs.

Corpora Quadrigemina. Oculomotor paralyses; reeling gait; blindness (?); deafness (?).

Pons and Medulla. Crossed paralyses of face and limbs, or tongue and limbs. Other cranial nerve lesions.

Cerebellum. Marked cerebellar ataxia; marked vomiting. Often a latent region.

Base, Anterior Fossa. Mental impairment; disturbances of smell and vision; exophthalmus.

Base, Middle Fossa. Disturbance of vision; oculomotor disturbances; hemiplegia.

Base, Posterior Fossa. Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis. Disturbance of vision; oculomotor disturbances.

Continuous headache that resists treatment, persistent and intractable vomiting, optic neuritis, with a localized spasm or paralysis, make a combination of symptoms rarely presented by any other disease. The differentiation from abscess is given under Abscess. It is of great importance to determine whether or not the growth is syphilitic, as a syphilitic growth, a gumma, may be amenable to treatment.

FIG. 78.

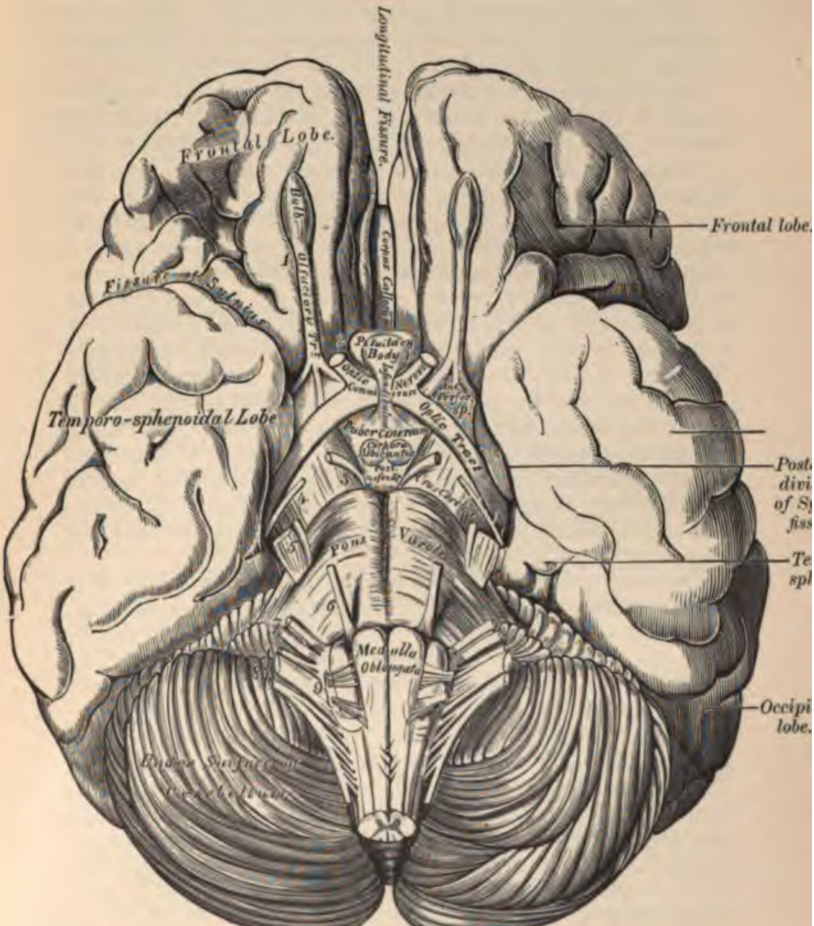


Fig. 78, reproduced from GRAY, will prove of value in the attempt to localize tumors of the base of the brain, and to explain the accompanying paralyses of cranial nerves.

MULTIPLE SCLEROSIS.

By multiple sclerosis is meant that diseased condition in which sclerotic plaques develop irregularly in the brain, spinal cord, and peripheral nerves.

The diagnosis of the disease is based very largely upon the existence of what is known as the **intention** or **voluntary tremor.** This may be regarded as almost pathognomonic. If a patient with multiple sclerosis attempts to perform any voluntary act, as, for example, the picking up of a pin from the table or the carrying of a glass of water to the lips, the hand is thrown into a tremulous motion, the tremor usually being a coarse one. Somewhat analogous is the **nystagmus** which is met with in these patients, particularly when the patient attempts to hold the eye steady, as the eyeball will quiver and move from side to side, constituting the well-known nystagmus. Somewhat similar, too, is the change in the manner of speech. There is an interruption of the continuous and smooth flow of speech, the patient separating words or even syllables, and being obliged to speak slowly and deliberately in order to enunciate the words distinctly. This is known as the **scanning speech.** The discovery of the intention tremor associated with nystagmus and scanning speech would be the discovery of multiple sclerosis.

The usual complaint of the patient is that of vertigo, occasional sensory disturbances in the arms and lower extremities, headache, general weakness, fatigue upon slight exertion. Epileptiform attacks may occur. The trembling before described, is noted, and gradually becomes more marked. In many cases, too, difficulty in walking is the result, not alone of the trembling, but of a spastic condition of the muscles induced, probably, by an invasion of the lateral columns of the cord. The reflexes may be very much exaggerated. Disturbances of the bladder and rectum may or may not occur. The cranial nerves may be involved and diplopia and amaurosis are met with. In some cases psychical disturbances are pronounced, and it is not unusual for patients to be looked upon by their friends as weak-minded.

The diagnosis in typical cases is extremely easy, but in the early stages may be impossible.

From *paralysis agitans* it can usually be separated, as in the former the peculiar gait, the muscular rigidity, and the fact that the tremor ceases upon volition enable us to recognize that disease. The exaggerated reflexes make it resemble oftentimes *lateral sclerosis;* but in pure lateral sclerosis there is not the intention tremor, the nystagmus, or the scanning speech. From *tabes dorsalis* it is differentiated by the absence in tabes of the intention tremor, of the nystagmus, of the scanning speech, and by the fact that in tabes the reflexes are lost. There are, too, in tabes, marked disturbances of sensation, notably the lightning pains and the visceral crises, as well as the characteristic changes in the pupil.

GENERAL PARALYSIS OF THE INSANE.

Chronic Diffuse Meningo-encephalitis.

General paralysis of the insane, commonly known as softening of the brain, is, in its pathology, very similar to tabes dorsalis, save that in general paralysis the brain is chiefly affected, while in tabes the cord is the seat of the chief lesion.

The causes of general paralysis are, first, syphilis, and, secondly, excessive bodily and mental overstrain, and occasionally traumatism. The mental symptoms are those usually first noted. In what is usually called the prodromal stage, it is seen that the patient is becoming forgetful, is absent-minded, makes mistakes in spelling or in speaking, and seems to think slowly. He becomes tired easily; his disposition changes. Previously good-natured, he may become ill-natured and cross. He grows careless about his dress, violates the ordinary proprieties, becomes indecent, obscene. It is noted, too, that in his speech he mixes syllables, uses occasionally the wrong word. He is bothered with insomnia. At this time, if the physician examines him, he may notice an inequality of the pupils, a clumsiness and awkwardness in the gait, an uncertainty and irregularity in the handwriting, and a fibrillary tremor of the tongue.

Following this prodromal stage comes the stage of **exaltation**, in which the patient is perfectly happy, imagining himself very rich, very learned, perhaps a ruler over an immense kingdom, or some important personage, as Washington or Napoleon. His memory for recent events is poor, but for the events of years past, oftentimes good. He does foolish things; spends money in a lavish manner; commits offenses against public morals or against the laws, for which he may be arrested.

Following this is the stage of **depression**, in which the patient is troubled by persecutions, and becomes hypochondriacal. "The lack of all power of criticism in these delusions and the inability to systematically liberate them, and the usual rapid course of this stage, distinguishes the general paretic from the insane patient." Gradually the patient lapses into a condition of mental impotency or of dementia.

Along with these psychic phenomena there are certain disturbances of motion and of sensation that must be mentioned. The pupils are usually inactive; there is a loss of the perception of pain. The tendon reflexes are frequently lost, though they may be exaggerated. Not infrequently, too, what are known as paralytic attacks occur, epileptiform seizures that may be followed by a comatose condition or by hemiplegia. During these attacks there is quite a considerable elevation of temperature. These attacks may be repeated frequently in the course of a day, or may occur only at long intervals. The disease usually lasts from one to six or eight years.

CEREBRAL SYPHILIS.

Syphilis, as it affects the brain, is a late manifestation. The syphilitic disease may be in the shape of a gumma, of an obliterating endarteritis, or of a diffuse chronic inflammation of the meninges and the underlying encephalon. The gumma is recognized by the symptoms already described under the head of tumor. There is vomiting, headache that is usually worse at night, choked disk, oftentimes paralysis of the cranial nerves. If the gumma presses upon the cortex, irritation is shown by convulsions that tend to recur, may be followed by paralysis, gradually extending as the tumor enlarges. Endarteritis presents the symptoms of cerebral thrombosis, that is, practically those of cerebral hemorrhage, but with a slower onset. Preceding the formation of the thrombus there are frequently nocturnal headache, dizziness, slight mental aberration.

The diffuse, chronic form of syphilis varies in its manifestations according to the extent of the disease process and its location. There may be loss of memory and intellectual power, tremor, ataxia, monoplegia. The symptoms are frequently identical with those of multiple sclerosis and of general paresis of the insane.

It need scarcely be said that in attempting to determine the nature of the cerebral difficulty, evidences of syphilis should be sought for wherever they might be found, as in an old adhesion of the iris, scars in the throat or over the tibiæ, or on the penis, enlarged glands, history of miscarriages, of alopecia, etc.

CHRONIC HYDROCEPHALUS.

Hydrocephalus, an accumulation of fluid in the ventricles of the brain, either an exudate or a transudate, may be congenital or acquired. The latter form is frequently associated with tuberculosis or follows some previous inflammation, as meningitis. A tubercular history is of value in establishing the diagnosis.

When it begins in infancy the child's head may enlarge quite rapidly. The fontanelles are large, the sutures gaping, the membranes bulging through the fontanelles; at times fluctuation or pulsation can be detected. The veins of the scalp are frequently swollen. Nystagmus is at times present. The child shows faulty mental development, does not learn to talk, is imbecile, may be blind, and is usually poorly nourished. The child learns to walk very late, if at all. There may be paresis or paralysis. The reflexes are frequently exaggerated. The condition is usually recognized readily, though it may be confused with rickets. But in rickets, while the head may be deformed, it is usually more box-shaped, and there are evidences of deformities in other bones, as in the lower extremities, the rachitic rosary, etc., and in rickets, blindness, paralysis, mental weakness are not found. The rachitic child is unusually bright.

MÉNIÈRE'S DISEASE.

Ménière's disease is a paroxysmal vertigo, supposed to depend upon disease of the internal ear or of the semicircular canals. It is usually associated with partial or complete deafness of one ear. At times there has been noted perforation of the drum with a history of suppurative middle-ear disease. Tinnitus aurium may be constantly present. The dizziness is usually paroxysmal. The patient may have few or many attacks daily. There is ringing in the ears, a feeling as though objects in the room were falling or whirling about, a sense of weakness and faintness, at times nausea; patients sometimes fall to the ground. This may pass away and the patient may feel perfectly well until another paroxysm comes on. In some cases the vertigo is only noted in particular positions, as when the patient lies upon the right or left side, or when he sits erect. The deafness, the paroxysmal character of the vertigo, failure to find any disease of the cerebellum or other portions of the nervous system, make the diagnosis usually simple.

GENERAL AND FUNCTIONAL NERVOUS DISEASES.

CHOREA.

Chorea, or St. Vitus's dance, is a disease in which involuntary, irregular movements occur, usually beginning in the upper extremities and the face, where they are most marked, though not infrequently involving the lower extremities or the entire body. Only the voluntary muscles are attacked. When one-half of the body is involved the disease is spoken of as hemichorea.

In a fully developed case in a child, for children (girls) are oftenest attacked, the child is seen to be in almost constant motion. The muscles twitch, the arms are extended and flexed, adducted and abducted; the fingers are never quiet, and the same restless movements are observed in the muscles of the lower extremity, and sometimes in those of the neck and face and the trunk. In chorea, movements cease entirely when the patient is asleep. These children, when the disease is well advanced, have great difficulty in walking. They are unable to handle a pen, and may also be unable to carry food to the mouth by means of a spoon or a fork. Dressing may become impossible. The more the attention of the bystanders is attracted to the patient, the more difficult it is for her to perform voluntary acts. These choreic patients are usually quite anæmic. Sensation is not altered. The reflexes are usually preserved. The mental condition of the patient may be unchanged, though at times children become irritable, cross, peevish, or somewhat dull and stupid. The disease usually lasts for several weeks, and shows a tendency to spontaneous recovery. In pregnant women, however, chorea is a serious affection.

The frequent occurrence of endocarditis and myocarditis with chorea has been often noted; and in chorea, too, inflammation of the joints, usually looked upon as rheumatic, is of common occurrence. Whether the rheumatism is to be looked upon as the exciting cause of the chorea and of the endocarditis, or whether all three conditions are due to some infectious organism not yet isolated, is still a question.

One can scarcely go astray in the matter of diagnosis when one notices the irregular, involuntary movements of the upper extremities, movements that cease during sleep, that are accompanied by anæmia, oftentimes by valvular murmurs and by rheumatism. The movements of athetosis do not cease during sleep, and there is usually a history of infantile cerebral palsy. The tremors of paralysis agitans, of multiple sclerosis, can scarcely be confused with those of chorea, if careful examination be made for evidences of these diseased conditions. The exciting cause of chorea, too, can sometimes be discovered in a sudden fright, and occasionally chorea seems to be due to imitation. Thus one child sees another child with chorea, and, through imitation, performs the same irregular involuntary movements.

HEREDITARY CHOREA OR HUNTINGTON'S DISEASE.

Hereditary chorea is a rare affection, making its appearance usually between the twentieth and fortieth years. The onset is gradual, the choreic movements being at first of mild intensity, but gradually in the course of months or years becoming more and more marked. Finally all the voluntary muscles are involved, including those of the face and of the tongue. This causes the patient to have a peculiar, uncertain gait, and difficulty in speech. The face, too, is thrown into marked contortions and grimaces. During sleep, and sometimes by the exertion of strong will-power, the movements cease; the muscular strength is undiminished. There are very rarely alterations in the reflexes or in sensation. Mental weakness, apathy, and even hypochondriasis, are not uncommon. The prognosis is unfavorable, the disease pursuing a chronic, progressive course.

The diagnosis is made on the late appearance of the chorea and the history of heredity. Usually the patient is able to trace back the disease through several generations. The fact, too, that the movements become gradually worse in spite of treatment, affords aid in diagnosis.

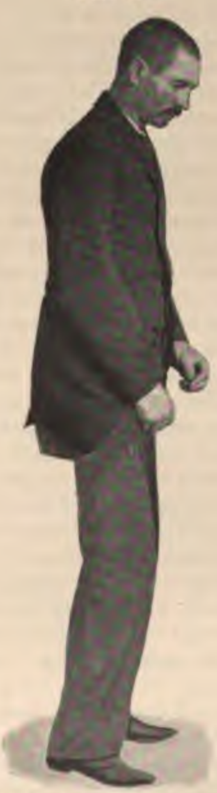
PARALYSIS AGITANS.

Paralysis agitans is a disease of old age, oftenest appearing after the age of fifty. Various etiological factors have been recognized, but there is no common cause for the malady. Fright, anxiety, mental worry, infectious diseases, hereditary neurotic taint, all seem to have some influence. The condition is recognized by (1) the tremor; (2) the weakness and rigidity of muscles; (3) the difficult and slow muscular movements; and (4) the peculiar gait.

Usually there is first noticed a **weakness** in the extremities, soon followed by a **tremor**, which often appears in the right arm. This gradually extends to other parts of the body, and it is then noticed that there are uniform vibratory movements, the oscillations being few in number, rarely exceeding four to the second. This tremor interferes with the use of the hand in writing. It does not cease when the patient goes to bed, and in fact may become most intense just before the patient drops off to sleep. The muscles of the patient are not only weak, but acquire a certain **rigidity**. As a result, there is a peculiar attitude of the patient which, with his *gait*, often enables the diagnosis to be made at a glance. The head is bowed forward, the whole trunk is somewhat bent; the arms are slightly flexed, are held close to the trunk, and the thumb is adducted and held in apposition with the fingers, the hand being in a position it would assume in writing. As the patient walks he seems to have a tendency to fall forward, and takes short, quick, almost running steps to avoid this. This is known as the propulsion or **festination** of paralysis agitans. The rigidity of the muscles gives to the face a peculiar wax-figure-like appearance. The voice may become shrill as that of an old man. The reflexes are normal. There are no sensory disturbances. The bladder and rectum show no alteration in function. Occasionally sensations of heat are complained of, and hyperidrosis has been noted.

The disease, from the tremor, may be confused with Graves's disease or with multiple sclerosis. In **Graves's disease**, however, the cardiac irritability, the exophthalmus, the goitre, the more rapid tremor, enable the diagnosis to be readily made. **Multiple sclerosis** is recognized by the fact of its beginning at an earlier period in life, by the tremor ceasing when the patient is at rest, and becoming violent when voluntary movements are attempted; by the lack of muscular rigidity and contracture; by the presence, oftentimes, of nystagmus, affections of the optic nerve, disturbances of the bladder and rectum. The peculiar position of the patient as he walks, and his hurried, festinating gait also enable us to recognize a patient with paralysis agitans.

FIG. 79.



Paralysis agitans. (LYMAN.)

HYSTERIA.

No organic lesion has yet been found to explain hysteria. The nervous system seems to be in a condition of increased sensitiveness; slight external or internal impressions produce unusual effects, both upon the brain, the cord, and the peripheral nerves. Only the main facts in the diagnosis can here be given.

The disease is met with oftenest in young girls, though children, adults, and men are affected. Usually a neurotic family history is obtainable. Frequently, too, there are faults in the education of the child who becomes hysterical. These patients are excitable, changeable in temperament, readily frightened, always thinking of themselves. They are easily excited to hysterical laughing or crying fits, alternately laughing and sobbing, partially losing consciousness, particularly when sympathy is expressed for them by their friends. Other hysterical seizures may occur. There may be violent spasm of the laryngeal muscles; there may be hysterical convulsions resembling those of epilepsy. The **hysterical convulsion** is often excited by some quarrel or some sudden fright, but in this convulsion the patient does not usually lose consciousness completely, as in epilepsy; does not bite her tongue; the convulsion lasts longer, and during the convulsion the patient is apt to laugh and cry, or scream.

Many of the cranial and spinal nerves may be affected in hysteria. Thus, there are found alterations in the sense of smell and of hearing; disturbances in vision, as, for instance, loss of the ocular sense and contraction of the field of vision, are not infrequent. The taste may become perverted; neuralgic pains in the distribution of the trigeminus may be noted. The scalp is sometimes exquisitely tender. The hysterical spasm of the glottis has already been spoken of. The patients sometimes are suddenly unable to speak above a whisper. This hysterical aphonia may last for some minutes or for many weeks, and as suddenly disappear. Anæsthesia of the mucous membrane of the fauces is so common as to be a valuable aid to diagnosis. Variations in the rapidity of the heart's action and of respiration are not uncommon. The globus hystericus is another very common phenomenon, probably due to a spasmodic contraction of the œsophageal muscles, giving rise to a sensation as though there were a lump in the throat. In hysteria the abdomen may be enormously distended with gas (hysterical meteorism), and may resemble the tympanitic abdomen of typhoid, or even the abdomen of a pregnant woman. At times it is necessary to administer an anæsthetic in order to determine the nature of the abdominal distention. Vomiting is very common in hysterical girls.

Sensory and motor disturbances are among the commonest phenomena of hysteria. Hemiplegia and hemianæsthesia have been noted. Hysterical paralysis usually appears suddenly, and may disappear as suddenly. These paralyses are oftentimes irregular in distribution and are usually of the flaccid type. Frequently the paralysis is not complete. Muscular spasms, oftentimes of single muscles, are noted. The joints may be contracted, and it is characteristic of the hysterical contracture that it occurs suddenly. Under chloroform the hysterical contracture disappears. Anæsthetic areas are sometimes found involving half the body, one limb, or irregularly distributed patches. An anæsthesia that involves the entire body may be set down as hysterical. Hyperæsthesia over circumscribed areas is not infrequently seen, and ovarian hyperæsthesia is looked upon as almost pathognomonic. Pains in the joints are not infrequent. The urine of hysteria may be very scanty, but is often, particularly after a fright, or after a hysterical seizure, of increased amount, pale, and of low specific gravity.

The diagnosis of hysteria is to be made by excluding all other diseases. We are aided in making the diagnosis by a knowledge of the previous attacks, by a knowledge of the neurotic taint in the family or in the individual. The symptoms are also without order, very changeable, and there is no physical basis for the trouble to be discovered. Ovarian tenderness, tenderness over the point of exit of the intercostal nerves, pharyngeal anæsthesia, and globus hystericus, are valuable aids to the diagnosis.

EPILEPSY.

Epilepsy is a disease characterized by a condition of unconsciousness combined with convulsions. Oftentimes the predisposing or exciting cause can be found in heredity, in chronic toxæmia, as with alcohol or lead, in the acute infectious diseases, notably scarlet fever, in syphilis, and in injuries of the head. Reflexly, epilepsy is due in some cases to injuries or irritations of the peripheral nerves, as in diseases of the nose, the stomach, laryngeal polypi, foreign bodies in the ear, an inflamed nail-bed (Hirt). Fright and other psychic disturbances are assigned as causes in certain instances. It usually makes its appearance before the twentieth year.

Clinically epilepsy is divided into major epilepsy or *grand* *mal*, minor epilepsy or *petit mal*, and the epileptic **equivalents.** In a large proportion of cases, preceding a genuine epileptic attack there is some peculiar sensation which the patient learns to regard as a warning or premonition. This is known as the *aura*. The auræ have been divided into **psychic** and **somatic** auræ. Among the *psyschic* auræ and the auræ involving the nerves of special sense should be mentioned an irresistible desire on the part of the patient to run, a sudden reminiscence of bygone days; vertigo, headache, peculiar odors, tastes, sounds, and sudden flashes of light. The *somatic* auræ may be *motor*, as the twitching of a certain muscle or muscles, or the sudden contracture of the same, strabismus, spasm of the glottis, vomiting; or *sensory*, as sensations of tingling, of burning, of numbness; or *vasomotor*, as, for instance, sudden pallor of the skin with sudden coldness of the hands and feet, blushing, and sweating. It is characteristic of the aura that it is always centrifugal in its propagation. It always seems to the patient as though the peculiar sensation or contraction was creeping upward, slowly or suddenly, toward the head. Patients sometimes learn that by interrupting this creeping process, as by firmly grasping the arm in which the aura is felt, by rubbing it briskly, or by binding it with a cord, an attack can be interrupted.

The aura is sometimes so far in advance of the attack that the patient is able to lie down in a place of safety before the attack really comes on. In other instances the time of warning is too short, the patient falls suddenly to the ground with a cry, the head is thrown back, the fingers are clenched, the thumb adducted, and the patient is in a condition of **tonic spasm.** The muscles of respiration cease to act, so that the face becomes cyanotic. The eyeballs protrude, the pupils are dilated. Then the second period of *clonic spasm* begins. The muscles of the face, of the hands, of the extremities are thrown into violent and somewhat irregular energetic movements. Often the tongue, getting between the teeth, is bitten. The foam which covers the patient's lips will then be stained red. The pulse becomes rapid; the body may be covered with perspiration. Cyanosis is now relieved as the muscles of respiration again perform their function. The stage of tonic contraction usually lasts from a few seconds up to half a minute; the stage of clonic contraction from one minute to five. During an attack the temperature may rise slightly; the reflexes are lost, including the conjunctival and pupillary reflexes. The urine, the feces, and the semen may be involuntarily passed.

Following an attack the patient usually passes into a deep sleep resembling coma. The urine may be albuminous. The patient's mind is often somewhat cloudy; for some hours following the attack he complains of headache, is slightly bewildered, and may have some trouble in talking fluently.

These attacks may occur at long intervals, or in severe cases may be repeated frequently during the course of the day. Where the attacks are frequently repeated the temperature may rise to a high point, the patient becomes comatose, and in a condition known as the *status epilepticus.*

The minor epilepsy, or *petit mal,* is a form of epilepsy where the attack consists of a transient loss of consciousness without marked spasm. The countenance may be observed suddenly to become pale; the patient has a staring look. There may be a very slight twitching of the lips or of the tongue. This soon passes away, and the patient, entirely unconscious that anything has occurred, resumes his conversation or his writing, or his ordinary work, just where it was interrupted by this peculiar attack.

Some patients with epilepsy retain their ordinary intelligence to the last. Where, however, the attacks occur frequently the patient's mind is usually altered; the child becomes cross and irritable, somewhat dull and stupid, and may end his days in the insane hospital or the asylum for feeble-minded.

The differential diagnosis, where there is a history of repeated attacks, is usually easy. It is well to follow the rule of Oppenheim, never to make a diagnosis of epilepsy upon the ground of a single attack. Simple *syncope* may resemble somewhat epilepsy, but in this condition—anæmia of the brain—there is marked pallor of the countenance. The pulse is extremely feeble. There is a lack of the convulsions, of the involuntary discharge of urine and feces. *Hysterical* convulsions are distinguished by the known hysterical character of the individual, the fact that the tongue is rarely bitten, that the patient talks and cries during the convulsion, assumes various unusual and striking attitudes, and that the pupillary reflex is preserved. The convulsions that occur in the course of paralytic dementia, the convulsions of tumor of the brain, syphilis of the brain, nephritis, etc., need only to be thought of, to enable us by investigation to arrive at a correct diagnosis.

Nocturnal Epilepsy, the form where the convulsions occur only during the night, may often be recognized by the injury to the tongue, the headache and dulness in the morning, the discharge of urine or of feces during sleep. Those sleeping near by are often aroused, too, by the epileptic cry, or by the noise made by the patient during the convulsions. In some cases *malingerers* simulate epilepsy, but malingerers rarely bite the tongue, the cyanosis is never so marked, the pupillary reflex is preserved, and there is not the post-epileptic bewilderment and coma.

Jacksonian Epilepsy is a term applied to a partial epilepsy having its origin in cortical irritation, as from the pressure of a spicule of bone, from a tumor or an abscess. In Jacksonian epilepsy a single muscle or a group of muscles, as, for instance, those of one arm, may be thrown into a spasm, but consciousness is rarely lost. The patient may, in fact, watch the spasm in the arm. This attack may last some little time, may be repeated within a few days, but shows a tendency to become more and more diffuse, that is, more muscles are involved with each subsequent attack. Sensory disturbances are not uncommon in the part involved. Later, the part usually becomes weak, or even completely paralyzed. Firm pressure over the affected area may cause severe pain or an epileptic seizure. Jacksonian epilepsy is the form of epilepsy that is oftentimes relieved by surgical operation.

HYSTERO-EPILEPSY.

Hystero-epilepsy is a term applied to designate a condition in which the patient, usually a woman, suffers from attacks that seem to be a mixture of hysteria and epilepsy, or at least of epileptiform convulsions. The epileptiform attack may begin quite suddenly, the respiration stopping, the patient falling to the ground, being seized with convulsions and contractions of almost all the voluntary muscles. But, in contrast to the true epileptic convulsion, she does not have the clonic spasms, nor pass into a condition of stupor or coma, but she now assumes strange positions, bends the body in the form of an arch, laughs, cries, howls. Then follow hallucinations; she assumes positions indicative of anger, of laughing, of pain; she may even become delirious. During this attack anæsthesia may be marked. The patients will sometimes, following an attack, remember what has occurred, at other times they do not. A valuable aid in diagnosis is afforded by the fact that deep pressure over the ovarian region cuts short the attack.

The differential diagnosis between true epilepsy and hystero-epilepsy is in some cases rather difficult. In hystero-epilepsy, however, we have a patient of evident neurotic temperament. There is rarely an aura; while the patient may froth somewhat at the mouth, she rarely bites the tongue. The convulsion is longer, often lasting ten or fifteen minutes; the epileptic convulsion seldom lasts longer than five minutes. While the tendon reflexes may be lost, the pupillary reflex responds to light, while in epilepsy this is not true.

TRAUMATIC NEUROSES.

Closely allied to hysteria are those peculiar affections known as the traumatic neuroses. In these cases there is usually a distinct psychic and bodily shock. The disease is met with so frequently following railway accidents that it is often spoken of as "*railway spine.*"

The symptoms may come on immediately after an accident, or may be more gradual in their appearance. The mental symptoms show the patient nervous, anxious, with the attention almost constantly directed to himself and to his deplorable condition. He may become morose, melancholy, and despondent. Motor disturbances are shown by the weakness or paresis of certain muscles, usually those of the lower extremities. There is complaint of pain in the head and back. Areas of hyperæsthesia can generally be found; particularly is the skin extremely sensitive over the spine, which the patient is always sure has been injured. In other cases patches of anæsthesia may be found, and paræsthetic sensations are frequently complained of. The reflexes are variable. In some cases disturbance of vision, as, for instance, contraction of the visual field, is noted. While the railway spine is to be looked upon as a functional trouble of a nature akin to hysteria, it must not be forgotten that in some of these cases true organic disease of the cord or of the brain manifests itself later. It is a remarkable fact, too, that the condition of the patient oftentimes improves quite suddenly when the litigation over the accident has been settled.

The history of an injury, the impending litigation, the mental anxiety or despondency, the atypical distribution and combination of motor and sensory disturbances, corresponding to no known organic disease, the localized hyperæsthesia over the spine at the supposed point of injury, all go to make up a clinical picture quite characteristic of this still rather mysterious affection.

NEURASTHENIA.

Neurasthenia is a condition of nerve weakness in which no pathological lesion is recognized during life or after death. The symptoms are in many cases general; in others local, so that sometimes a general neurasthenia is spoken of; in other cases a cerebral, a spinal, a gastric, or a sexual neurasthenia.

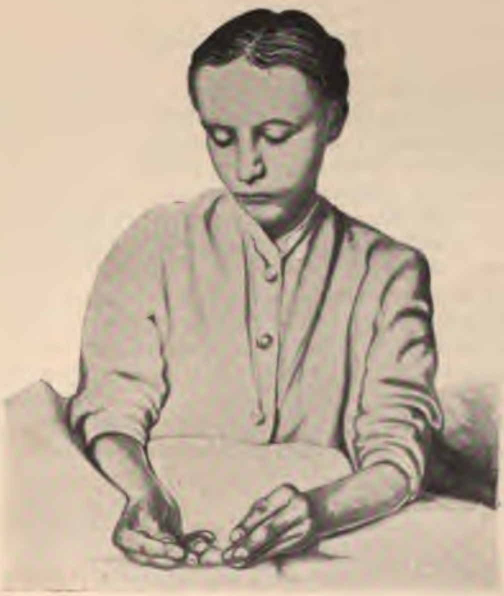
Psychic disturbances are usually pronounced in neurasthenics. They worry a great deal, are imaginative, may be morose, hysterical, hypochondriacal. They are easily frightened. Pains are usually complained of in some part of the body. It may be there is a severe headache, or that the back is tender, or there is a tenderness in the ovarian region. Flushes of heat, feelings of numbness, are frequently noted. The digestion is much disturbed, the appetite being capricious, and there being after a meal a sense of weight and heaviness in the stomach, perhaps attended by bloating, eructation of gas, or even by vomiting. The bowels are often obstinately constipated. The sexual function is frequently abnormal, in men particularly, impotence being noted. The urine of neurasthenics often shows an increase in the urates and oxalates. Sleep is disturbed, or the patient suffers from pronounced insomnia. The hands and feet are cold; the pulse is often feeble; the heart may be irritable. In severe cases the patient becomes emaciated, the mental condition markedly changed, and the patient so weak as to be bedridden and truly in a pitiable condition.

The diagnosis is made chiefly by exclusion of any organic disease. Particularly must we exclude organic diseases of the stomach, of the brain, *e. g.*, tumor and paralytic dementia, and of the spinal cord. From hysteria the disease is not always readily differentiated.

TETANY.

Tetany is a rare disease, characterized by paroxysmal, tonic muscular spasms. These spasms may be preceded by painful sensations or paræsthetic feelings in the fingers and arms, or may come on suddenly. The flexors of the fingers and of the wrist are usually affected. The fingers are drawn together with the thumb adducted, the hand, as Trousseau described it, being in the shape the obstetrician gives it when introducing it into the vagina. The affection is usually bilateral. The elbow may be slightly flexed, the arm adducted. In some cases the feet are affected, the toes being flexed, the big toe often drawn under the second and third, and the foot assuming the position of plantar flexion. The spasm may last a few seconds, minutes, or hours. The disease is unattended by loss of consciousness or by fever.

FIG. 80.



Tetany. (OPPENHEIM.)

The diagnosis is made from the peculiar position assumed by the fingers and toes, and the paroxysmal nature of the spasms. Trousseau's sign is of great value in diagnosis. He found that by pressure upon the larger arteries, as, for instance, the brachial, the spasm could be artificially excited. Erb has also proven that there is an increase in the electrical as well as in the mechanical excitability of the nerves. In some cases, stroking the face gently with the fingers, produces vigorous contractions in the facial muscles.

The cause of the disease is still unknown, though it has been noticed oftenest in women, particularly those who are nursing children. It has also been seen following the infectious fevers, and in those whose occupation obliges them to use the arms and hands, as seamstresses, telegraph operators, washerwomen. The disease runs a favorable course, though it may seriously interfere with the occupation of the patient. If not alone the hands and feet are involved, but other muscles of the body as well, it may become an extremely serious affection.

THOMSEN'S DISEASE.

In 1876 Thomsen described a disease that had been noticed in his own family for five generations, to which the name *Thomsen's disease* or *myotonia congenita* has been applied. This affection is characterized by a painless tonic spasm in the muscles during voluntary movements. Not alone may the movements of the arms or of the legs, as in running, gymnastics, be impossible, but the muscles of the tongue may also be involved and speech disturbed. No sensory disorders are noted. The muscles are usually well developed and show an increased excitability to the galvanic current. The disease does not shorten life, though it may last for many decades. The nature of the affection is still unknown, though heredity plays an important part in its etiology. It is not yet fully decided whether the disease is not to be regarded more as an affection of the muscles than of the nervous system.

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